ARCHIVES OF OPHTHALMOLOGY.

THE HISTOLOGIC FINDINGS IN A CASE OF TUBERCULOUS CYCLITIS, AND A THEORY AS TO THE ORIGIN OF TUBERCULOUS SCLERITIS AND KERATITIS.[1]

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(From the Pathological Laboratory of the Massachusetts Eye and Ear Infirmary.)

(*With twelve figures on Text-Plates XII.–XIV.*)[2]

CASES of episcleritis, scleritis, and sclero-keratitis, with nodule formation, are generally classified according to etiology into two groups—one group comprising the cases of rare occurrence due to syphilis, leprosy, or active tuberculosis, and the other those of frequent occurrence due to some obscure cause, commonly supposed to be rheumatism. The second group is typified by a distinctive clinical condition designated anterior nodular scleritis. In a previous communication[3] I have brought forward evidence to show that the latter condition is always due to tuberculosis. This evidence, while partly histologic, consisted chiefly in the results of subcutaneous tuberculin injections in a consecutive series of cases. Since then I have confirmed my observations

[1] Read at the meeting of the American Ophthalmological Society, Washington, May, 1910.

[2] The photomicrographs were made by the Pathologic Department of Harvard University.

[3] "Tuberculous Scleritis, a Commonly Unrecognized Form of Tuberculosis," *Boston Med. and Surg. Jour.*, March 14, 1907, p. 317.

in a still larger series of cases, and in every case have obtained a general reaction, and in most cases also a local reaction, to the tuberculin test. While a local reaction in the eyes did not always occur in the diagnostic tests, it could practically always be obtained later if treatment with old tuberculin was carried out with this end in view. Local reactions repeatedly obtained in this way are, I am convinced, desirable, contributing, as I have elsewhere maintained,[1] largely to the success of the tuberculin treatment, provided sufficiently long intervals are allowed to elapse between them.

I have also pointed out that no sharp distinction should be made between cases of apparently pure tuberculous keratitis and cases of sclero-keratitis, that they both represent the same type of chronic tuberculosis, and that, in fact, most often, if not always, tuberculous keratitis has been preceded by scleritis. In addition, I have called attention to the fact that, like tuberculous scleritis, tuberculous keratitis has characteristic clinical features which serve to distinguish it from other forms of interstitial keratitis.

My experience in connection with ocular tuberculosis has been sufficiently extensive to justify the belief that the failure of certain observers to obtain equally consistent results with the tuberculin test in cases of anterior nodular scleritis has been due either to some error of technic or procedure in making the test, or to an erroneous clinical diagnosis. Probably the condition that is most often mistaken for nodular scleritis or sclero-keratitis is the neuropathic conjunctivitis recently described by me.[2] This occurs under the same conditions as neuropathic keratitis—most commonly under those which give rise to facial herpes, with which, in fact, it is often associated. Gumma of the ciliary body and sclera, judging by the few cases I have seen of this rare condition, should not readily be mistaken for tuberculous scleritis. Leprous scleritis is, of course, too rare to give rise to any confusion, and in any case its nature would, no doubt, be rendered clear by the presence of other manifestations of leprosy.

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[1] "Some General Considerations Concerning Tuberculosis of the Eye," *Ophthalmic Record*, December, 1908.

[2] "Neuropathic Keratitis and Some Allied Conditions," *Jour. Amer. Med. Assoc.*, July 17, 1909.

To Illustrate Dr. F. H. Verhoeff's Article on "The Histologic Findings in a Case of Tuberculous Cyclitis, and a Theory as to the Origin of Tuberculous Scleritis and Keratitis."

Fig. 1.—Photograph of anterior portion of globe after fixation in formalin, showing corneal opacities of old sclerokeratitis.

Fig. 2.—Same specimen seen from behind, showing punctate tuberculous exudates on surface of ciliary body. Note the conglomerate character of the larger exudates.

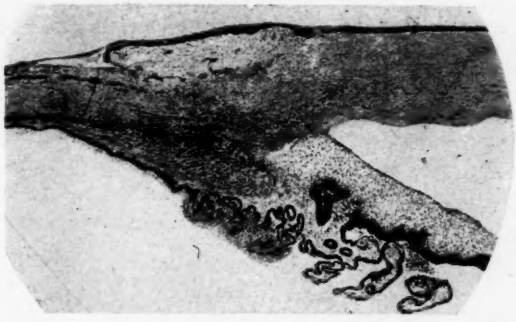

Fig. 3.—Section of one of the ciliary exudates. Its origin from the ciliary processes and its conglomerate character are well shown. Note also the character of the adhesion of the iris root to the ligamentum pectinatum and the vascularization of the sclera and cornea leading from the ligament. (Photo. × 18.)

Fig. 4.—Same ciliary exudate under higher magnification. Note that the overlying ciliary body is normal. (Photo. × 53.)

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Admitting that anterior nodular scleritis and sclerokeratitis are due to tuberculosis, the questions arise why such resistant and relatively avascular structures as the sclera and cornea should be the favorite sites of ocular tuberculosis, and how it is that the infection reaches them. The experimental observations of Stock[1] suggest that tuberculous scleritis is due to extension from a metastatic focus in the uveal tract. This apparently satisfactory explanation, however, is not supported by the results of a number of histologic examinations which showed the ciliary body practically normal. It is also not borne out by clinical observations, which, on the contrary, go to show that cyclitis is a late occurrence in scleritis, and apparently secondary to the latter. The experiments of Stock were made under highly artificial conditions—conditions that more resemble those obtaining in miliary tuberculosis than in the chronic tuberculosis of scleritis. Moreover, in the two animals in which Stock actually obtained scleritis, the latter obviously can be explained equally well by the theory that I shall bring forward in this paper. This theory was suggested by the following case, which, by its exaggeration of certain features possibly unusual, pointed to the explanation of features not unusual. The case also afforded an exceptionally good histologic picture of both the late and early stages of tuberculous scleritis and keratitis.

Frances F., female, aged thirty-four, admitted to the Massachusetts Charitable Eye and Ear Infirmary October 14, 1908. Fairly well nourished and general health good. Highly nervous temperament. Previous health good except that patient stated that she had pleurisy (?) ten months ago. She also stated that her left eye was affected similarly to present attack four and one-half years ago, and that the vision afterward remained much impaired. The present attack began two months ago, the left eye becoming increasingly red and painful.

On examination the right eye was found apparently normal, and with normal visual acuteness. The left eye showed near the limbus above two small, reddish-yellow nodules, typical of tuberculous scleritis. There was only moderate circumcorneal injection, intensified at the sites

[1] "Tuberkulose als Ätiologie der chronischen Entzündungen des Auges und seiner Adnexe, besonders der chronischen Uveitis," *Graefe's Arch. f. Ophthal.*, 1907, lxvi., p. 1.

of the nodules. The cornea showed old interstitial gray opacities, most marked laterally, where they extended out from the limbus on both sides. There were apparently no active foci in the cornea, the opacities evidently being due to old sclero-keratitis. The iris showed a few old posterior synechiæ, but was little inflamed. Visual acuity of left eye $\frac{1}{60}$. General physical examination negative.

Atropin solution was instilled into the eye, and the temperature taken every two hours for forty-eight hours as a control preliminary to the tuberculin tests. The temperature was constantly within normal limits. Koch's old tuberculin, 1*mg* was then injected subcutaneously, followed by no reaction in forty-eight hours. Ten *mg* were then injected. This was followed by a febrile reaction of 101° F., a reaction at the site of the injection, and a local reaction in the eye consisting in increased congestion and pain. After lasting several days the local reaction in the eye gradually subsided, leaving the eye slightly improved. October 31, 1908, the patient was discharged.

After leaving the hospital the patient attended the outpatient clinic, where an attempt was made to carry out the usual antituberculous treatment—forced feeding, rest, fresh air, and tuberculin injections. This was found difficult, owing to the nervous temperament of the patient, and her lack of pecuniary means. Nevertheless, she gained steadily in weight and the eye slowly improved. Koch's old tuberculin was injected once a week, or once in two or three weeks in case a reaction resulted from the previous injection. The doses varied from 1 to 3*mg*, an attempt being made each time to regulate the dose, so as to produce a slight reaction. As a result, slight local reactions in the eye were obtained several times, thus confirming the diagnosis. After five months' treatment the eye was free from inflammation and the patient was allowed to go to her home, which was at a considerable distance from the city.

Six months later the patient returned to the hospital, stating that about two months after discontinuing the tuberculin treatment her eye had again become inflamed, and that recently it had become excessively painful. There was very slight ciliary injection, and no nodules of scleritis. On account of the old corneal opacities it was difficult to determine whether or not there was active keratitis or iritis. The tension was markedly lowered, and the visual acuteness reduced to the counting of fingers at two feet. At the urgent request of the patient, who, owing to the pain and other circumstances, did not desire to undergo another course of prolonged treatment, the eye was enucleated by Dr. Savage, September 17, 1909. When last heard

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Fig. 5.—Small punctate ciliary exudate on *pars plana*, showing origin from subepithelial focus containing a giant-cell. (Photo. × 146.)

Fig. 6.—Showing vascularization and scarring of sclera about the filtration angle at site of old scleritis. Note that the ciliary body is normal. (Photo. × 30.)

Fig. 7.—Section stained by van Gieson's method, showing scarring and vascularization of sclera leading from ligamentum pectinatum. (Photo. × 40.)

Fig. 8.—Active scleritis extending from ligamentum pectinatum. Active foci are present in the ligament in other sections of this series. The underlying ciliary body is normal, except for a slight infiltration with lymphoid cells opposite the scleral foci. Note the tuberculous exudate between the two layers of the *pars ciliaris retinæ* involving the *pars plana*. Serial sections showed that this had no continuity with the scleritis. Note the adhesion of the iris root to the ligament. (Photo. × 15.)

from, in February, 1910, the patient was in excellent health.

Pathologic Examination.

After remaining in 10% formalin several days, the eye was opened and photographed. After three months in the formalin it was placed in a saturated solution of potassium bichromate for two days, washed in water six hours, and embedded in celloidin.

The globe is distinctly quadrilateral in shape, and measures antero-posteriorly, 21.5*mm;* vertically, 20*mm;* horizontally, 21*mm*. The cornea, 11.5*mm* x 11*mm*, shows extensive old sclerosing keratitis at the limbus, and several opaque interstitial spots away from the limbus (Fig. 1). Three of these are well defined, one of which is especially opaque. The surface of the cornea is perfectly smooth. The anterior chamber is of normal depth. The pupil, 1.5*mm* wide, shows numerous posterior synechiæ. The root of the iris in general is free, but here and there adhesions to the ligamentum pectinatum can be made out, which, however, do not extend to the apex of the angle. The lens is *in situ* and apparently normal. The internal surface of the ciliary body shows 10 discrete opaque white areas, making a striking picture (Fig. 2). All these are slightly elevated and are situated on the ciliary processes except three, which are just behind them. The largest is 1.5*mm* in diameter; the smallest, 1.5*mm*. The larger ones appear to be conglomerations of several small ones. The largest ones are situated about 4*mm* apart. They do not correspond in position accurately to the corneal opacities. The choroid and retina are *in situ* and apparently normal. The optic disk is greatly swollen.

Histologic Examination.—Sections were made at close intervals, so that practically the entire anterior part of the eye was examined. Many sections were mounted serially.

Cornea.—The corneal epithelium and Bowman's membrane are everywhere intact and normal, except that the epithelium shows an insignificant infiltration with leukocytes. Descemet's membrane is also intact. The endothelium is normal except beneath some of the active corneal lesions to be described. Clumps of cells, chiefly large lymphocytes, are infrequently found adhering to the endothelium. The anterior one-fourth to three-fourths of the corneal stroma is, for the most part, normal. The posterior portion is vascularized, vessels being found in a median section from limbus to limbus (Fig. 9). The vessels occur chiefly four to five lamellæ anterior to Descemet's mem-

brane, and almost no vessels are found anterior to the middle layers of the cornea. In places where they extend out from the ligamentum pectinatum they are immediately in front of Descemet's membrane. The vessels are in general large, and although three or four smaller ones may occur together, they are usually widely separated from each other. The vessel-walls seldom consist of more than a single layer of endothelium. In this vascularized portion the stroma, except in certain places, does not depart greatly from the normal, the chief change being an increase in the number of corneal corpuscles. Even immediately around the vessels the stroma is, as a rule, little altered, the vessels simply pushing apart the lamellæ between which they lie. In areas, however, evidently corresponding to the corneal opacities, more marked changes are found, although these are not so great as the macroscopic appearance of the opacities would seem to indicate. In what appear to be the older opacities there is a decided increase in the number of fixed cells, but little disturbance of the lamellæ. Many of the cells here show a finely vacuolated or reticulated structure, no doubt due to the presence of fat-globules. Such cells are particularly noticeable about the blood-vessels. One small opacity of this kind is found just beneath Bowman's membrane; all the others are in the posterior half of the cornea.

In one large area, evidently a scar of more recent date, the corneal stroma is replaced by tissue which has little resemblance to the normal. Here there is found ordinary fibrous tissue, with an abundance of cells which are larger than the normal corpuscles. Many of these cells contain large vacuoles filled with transparent substance staining bluish in hematoxylin, probably mucin. The fibrous tissue is slightly less eosinophilic than the normal lamellæ. The appearance produced closely simulates that of a fibro-sarcoma. Behind this area, but not in continuity with it, there is a smaller area which is undoubtedly in an active stage (Fig. 11). This occurs immediately anterior to Descemet's membrane, is about ½*mm* in diameter, and situated 3½*mm* from the limbus. In this the corneal lamellæ are destroyed or so altered as not to be recognizable, and replaced by actively proliferating cells. The nuclei are large and lie in no one general direction. The cell outlines are not distinct; apparently the cell bodies are all continuous, forming a richly nucleated syncytium. There are no typical giant-cells of the Langhans type. The mucoid degeneration of the cells is more marked here than in the area just described. Intimately connected with the syncytium there is a newly formed ground substance which

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Fig. 9.—Showing vascularization of cornea and sclera, and punctate tuberculous exudate involving posterior surface of iris continued into a synechia. (Photo. × 10.)

Fig. 10.—Same iris exudate under higher power, showing giant-cell and origin from subepithelial tissue. (Photo. × 46.)

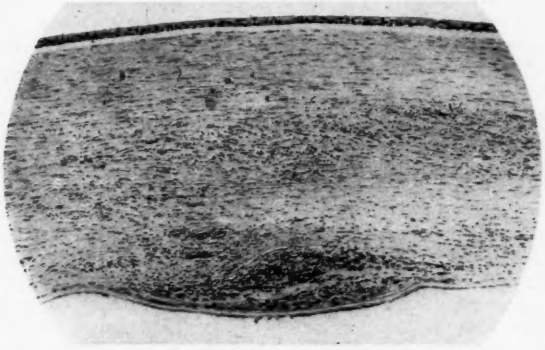

Fig. 11.—Active tuberculous focus in posterior layers of cornea, *keratitis punctata profunda*. Note proliferation of endothelium beneath it. In the middle layers of the cornea is a larger and older focus. (Photo. × 52.)

Fig. 12.—Showing papillitis, probably due to toxins carried along the central canal of the vitreous body. (Photo. × 14.)

is more strongly eosinophilic than the normal stroma, and which gives the reaction for collagen with van Gieson's stain. This area is free from blood-vessels. Behind it Descemet's membrane is normal, but the endothelium shows marked proliferative changes. Its nuclei stain more deeply, and it is lifted up in places by new cells which have formed beneath it. Newly formed endothelial cells are also being cast off into the anterior chamber. The cornea also shows several old scars, corresponding in size to this focus, which, no doubt, would represent the appearance of the latter when healed. At these places Descemet's membrane is greatly wrinkled, due, no doubt, to the contraction of the scar tissue. At the periphery above, in the region of the active scleritis, there is an active tuberculous process continuous with foci in the ligamentum pectinatum, which extends for a short distance between Descemet's membrane and the corneal lamellæ. Beneath this the endothelium shows changes similar to those beneath the active focus just described.

The Filtration Angle.—The ligamentum pectinatum is nowhere normal, but in some places it is more greatly altered than in others. Everywhere it shows an increase in its fixed cells, the nuclei of which stain more deeply than normal, and show other evidences of active proliferation. There is also infiltration of the spaces with chronic inflammatory cells. These cells, chiefly large lymphocytes, in places are also abundant, free in the angle, and, strangely enough, are about as abundant above as below. In some places the trabeculæ are not appreciably thickened, and the spaces of Fontana are well shown, but even in these situations small blood-vessels are almost always found in some of the spaces. In other places there is marked thickening of the trabeculæ, and almost complete obliteration of the spaces. Here the proliferation of fixed cells is excessive, and multi-nucleated cells are often seen. There are also large numbers of chromatophores. Some of the sections, which also include the foci of active scleritis, to be described, show still more definite tuberculous foci in the ligament, similar to the active focus in the cornea. The canal of Schlemm in all sections is much reduced in size and in many it cannot be recognized. This seems to be due partly to compression by the new fibrous tissue around it and partly to subendothelial proliferation of its own wall.

The filtration angle is practically never completely blocked by the root of the iris. Partial adhesions, however, occur in places. These are markedly different from those ordinarily seen in glaucoma. In some places delicate strands of iris tissue carrying blood-vessels bridge over the angle

and unite with the ligament. Most often, however, the adhesions are more definite, the iris stroma, as a whole, being drawn over and fused with the ligament. These adhesions are especially noteworthy from the fact that, instead of taking place at the apex of the angle, they occur at a considerable distance therefrom, terminating, however, at the beginning of Descemet's membrane (Figs. 3, 4, and 8). These are the adhesions noted macroscopically. From these adhesions the chromatophores of the anterior boundary layer of the iris sometimes have extended as a single layer of branching pigmented cells for a short distance over Descemet's membrane, replacing the endothelium. One set of serial sections shows one of these adhesions in an early stage of formation. It is seen, by comparison with a normal eye, that the adhesion is taking place at a point about opposite Schlemm's canal. It is seen also that the adhesion is not primarily due to inflammation of the iris, but to exudate and proliferated cells collected on the surface of the ligament having come in contact with the iris. By the contraction of this exudate it is evident that the close union of the iris to the ligament seen elsewhere would have resulted.

The Sclera.—In the region of the canal of Schlemm, and extending a variable distance backward, the sclera in places shows a marked increase in vascularization. The vessels are not only increased in number, but especially in size. The larger vessels consist simply of dilated spaces between the scleral fibres, lined by endothelium. These are, no doubt, veins. Some of the smaller vessels, evidently the arteries, show definite walls with muscular coats. Associated with this increased vascularization there is an alteration in the stroma of the sclera. The fibres are much distorted (Figs. 6, 7, and 9), are less eosinophilic than the normal sclera, and, in fact, present the appearance of scar tissue. In places these changes are limited to the region immediately about the ligamentum pectinatum, not extending farther backward than the iris root. In fact, they sometimes extend farther toward the cornea than backward in the sclera. The episcleral tissue at the limbus in those situations where the sclera is most affected is denser than normal, and vascularized with large vessels. Occasionally it also shows nodular infiltration with lymphoid cells.

In addition to the above changes, which are evidently the results of old scleritis, an active deep scleritis is found in one situation above (Fig. 8). As already mentioned, there is here an active focus in the ligamentum pectinatum, which extends also into the cornea. The vessels coursing backward from this region are surrounded by chronic

inflammatory cells, while the stroma shows foci of actively proliferating epithelioid cells. These foci, which are similar to the active focus in the cornea, occur not only at the same level, but at different levels in the sclera. The largest focus is in the middle layers of the sclera, over the orbiculus ciliaris. Beneath this there is a smaller focus which lies in the innermost layers of the sclera, and, by continuity, slightly involves the ciliary muscle. Serial sections show that there is here no continuity with any of the exudates on the surface of the ciliary body.

The Ciliary Body.—Except at the sites of the lesions seen in the gross specimen, and at the place just mentioned, the ciliary body is normal. In most places there is some exudation of lymphoid cells from the pars plana, but this is insignificant except in the vicinity of the gross lesions now to be described. These lesions are found to involve only the surface of the ciliary body, the underlying ciliary muscle being perfectly normal. They consist of sharply circumscribed exudates of actively proliferating epithelioid cells with relatively few lymphoid cells (Figs. 3 and 4). They lie, for the most part, upon the inner surface of the pigment epithelium, which is in most places intact. At the margins of the exudates the unpigmented epithelium may be seen passing over or into the proliferated cells and losing itself therein. Here it appears that the epithelioid cells of the exudate are largely derived by proliferation of the cells of the unpigmented epithelium. Closer examination, however, shows that in places the tips of the ciliary processes are broken through, and that it is from small foci just beneath the pigment layer that the cells of the exudate are chiefly derived. It is seen in these places that the proliferated cells raise up the unpigmented epithelium so that, in general, it may be said that the exudates occur between the two layers of the pars ciliaris retinæ. The epithelioid cells do not resemble discrete endothelial cells, but form a complicated, richly nucleated syncytium. In this, giant-cells of the Langhans type are occasionally seen. Upon the inner surfaces of these exudates, or rather upon the unpigmented epithelium which largely covers them, there is a smaller exudate of a different kind. This consists of elongated cells which extend along the fibres of the zonule. This exudate, on a small scale, appears to be identical with that generally seen in cyclitis which finally leads to the formation of cyclitic membranes. In some sections the conglomerate character of the larger exudates is apparent, separate foci having formed on adjoining ciliary processes and fused together.

Most of the exudates, as shown in Fig. 2, are situated

on the ciliary processes. Those situated on the pars plana, however, are of the same nature. In the smallest of these the origin of the epithelioid cells is especially well shown. In this one there is a small focus just beneath the pigment layer, consisting of epithelioid cells, among which typical Langhans giant-cells occur (Fig. 5). These cells have broken through the pigment layer and split the two layers of epithelium apart without anywhere breaking through the unpigmented layer.

The Iris.—Except for a slight infiltration with chronic inflammatory cells, most marked at the ciliary and pupillary margins and around the vessels, the iris stroma is not far from normal. There is, however, undoubtedly some increase in fixed cells. This is especially evident at the pupillary margin, where, in places, by cicatricial contraction, there is produced the beginning of ectropion uveæ. Extending out from the pupillary margin is a delicate membrane similar to that commonly seen in old chronic inflammatory conditions. This, however, does not completely block the pupil, because it is absent in the horizontal median sections. In one place there is a condition apparently analogous to the exudates of the ciliary body. A short distance from the pupil on the surface of the pigment layer there is a flattened nodule composed of epithelioid cells and an occasional giant-cell (Figs. 9 and 10). This is continuous, through a break in the epithelium, with a focus of similar cells just beneath the surface. This nodule appears to differ from those on the ciliary body only in the fact that it contains pigment cells derived from the pigment epithelium, which take the place of the unpigmented epithelial cells of the ciliary exudates.

Other Ocular Structures.—The lens is almost normal, showing only slight vacuolization of the cortex at the equator. The choroid is normal throughout. The retina, including the multipolar ganglion-cells, is also normal. The optic disk shows marked œdema. The condition is indistinguishable from the papillo-œdema associated with brain tumor (Fig. 12). There is very slight infiltration with lymphoid cells about the large vessels, and an equally slight exudation of these cells from the surface of the disk. This, however, is too insignificant to distinguish the condition from choked disk. The lamina cribrosa is curved slightly backward, but not abnormally so.

Numerous sections of the ciliary exudates and of the active foci in the cornea and sclera, stained and examined for tubercle bacilli, give negative results.

The clinical features of this case, which were typical of

recurrent tuberculous sclero-keratitis, and the local and general tuberculin reactions repeatedly obtained show conclusively enough that it was one of chronic ocular tuberculosis. No doubt had circumstances permitted the patient to have resumed the tuberculin treatment, the lesions would have again healed.

The punctate tuberculous exudates (Figs. 2 to 5) involving the surface of the ciliary body and its processes, so far as I can find, have not previously been observed. The most probable explanation for them is that they resulted from recent metastases which lodged in the superficial capillaries of the ciliary body. This is supported by the fact that the sources of the exudates were evidently the small subepithelial foci which were connected with them through breaks in the epithelium. It is certain that they did not result from the direct extension of the scleritis or keratitis, because the underlying portion of the ciliary body was normal. It is also clear that they were not metastases from the vitreous humor, since there was no primary focus from which they could have arisen. No one of the exudates could have served as a primary focus for the others, because all were in approximately the same stage of development, the larger exudates representing conglomerates of smaller.

The form of tuberculous cyclitis hitherto recognized has been an interstitial tuberculous infiltration of the ciliary body, occurring usually in continuity with scleritis, or, more rarely, as the active caseating form of tuberculosis. In the present case the condition might well be designated superficial punctate cyclitis. Such a condition, occurring in an otherwise normal eye, would obviously produce the clinical picture of serous cyclitis. As is now well known, the tuberculin test has shown that this clinical picture is undoubtedly sometimes due to tuberculosis. I would not imply, however, that this is usually the case. On the contrary, my own observations lead me to believe that serous cyclitis is often due to some cause as yet unknown.

The condition found in the iris in this case was clearly analogous to that in the ciliary body, and hence might properly be termed superficial punctate iritis. The small nodule (Fig. 10) was not observed clinically, probably owing to the

corneal opacities. It is evident, however, that, seen from in front, it would have the situation and appearance believed by Stock to be typical of chronic tuberculous iritis. This case, therefore, affords, histologic support for Stock's belief, which was based purely on clinical observations. In addition, this case suggests that chronic tuberculous iritis is associated more often than not with superficial punctate cyclitis.

The papillitis in this case was no doubt analogous to that which often occurs in association with other inflammatory conditions affecting the anterior portion of the eye. Van der Borg[1] has suggested that it is here due to toxins carried through the central canal of the vitreous, and thus concentrated upon the disk. It is noteworthy that in this case the swelling of the disk was due to pure œdema, practically unaccompanied by cellular infiltration or proliferation.

A condition of considerable interest was the extremely low tension the eye finally developed, although it was, of course, not uncommon. Not a sufficient area, apparently, of the surface of the ciliary body was involved to account for this, and there was no atrophy of the ciliary body. Moreover, in one of the cases carefully examined by van Sparwoude[2] in which there was low tension, the ciliary body is described as practically normal. Judging by the amount of cicatrization about the filtration angle, high tension would have been expected. This, however, was at no time noted clinically, and the facts that the retina was normal and the optic nerve neither atrophied nor cupped show that high tension could not have been present for any length of time. Two possible explanations suggest themselves: One is that, in spite of the obstruction in the ligamentum pectinatum and the canal of Schlemm, the excessive vascularization of this region permitted an undue outflow of aqueous humor. The other, which seems more probable, is that this excessive vascularization of the sclera, after it had become well established, reduced the capillary blood pressure of the ciliary processes and consequently the pressure of the aqueous humor.

[1] "Die Papillitis im Anschluss an Erkrankungen des vorderen Teils des Auges," *Klin. Monatsbl. f. Augenheilk.*, xlvi., 1908, p. 359.

[2] "Keratitis profunda scleroticans tuberculosa," *Academisch Proefschrift ann de Universiteit van Amsterdam*, 1909.

The active lesions in the cornea and sclera, consisting chiefly of small foci of epithelioid cells without caseation, were essentially similar to those described by other observers in cases of sclero-keratitis. On a smaller scale they appeared to be identical with those recently described by van Sparwoude[1] in three cases, and with those still more recently described in one case by Komoto. They were also similar to the small excised nodules from cases of scleritis which I have elsewhere described.

Failure, as in this case, to find tubercle bacilli in sections is, of course, not unusual in cases of chronic tuberculosis, but is none the less difficult to explain. It seems inconceivable that bacilli are not present in considerable numbers. Stock was usually unable to find them in his experimentally produced lesions, where they obviously must have been present. It, therefore, appears probable that in the tissues under certain conditions, doubtless having to do with immunity, they do not retain their characteristic staining reaction. A suggestion is given in regard to this by the exaggerated beaded forms often found in chronic lesions elsewhere, for it is not difficult to imagine that other bacilli must occur without even the beads, that are entirely unstainable by the usual methods. Animal inoculation was, unfortunately, not carried out in this case, owing to my absence from the city at the time the eye was removed. However, negative results are usually obtained after animal inoculation in cases of chronic ocular tuberculosis, owing, no doubt, either to the small dosage of bacilli or to the diminished virulence of the latter.

In regard to the source of infection in tuberculous scleritis and keratitis, the findings in this case lend no support to, in fact controvert, the view that it is through extension from foci in the uveal tract. On the other hand, it seems altogether improbable that scleritis is due to direct metastases from the blood. Against this is the fact that blood metastases to the sclera in cases of pyæmia, malignant tumors, and

[1] "Beitrag zur pathologischen Anatomie der Skleritis," *Klin. Monatsbl. f. Augenheilk.*, xlvii., 1909, p. 761. In this case the observer contents himself with the bare statement that tuberculosis was excluded. Just how, is left to the imagination.

miliary tuberculosis are practically unknown, if not entirely so. This is, no doubt, due to the fact that the sclera is poorly supplied with capillaries. There remains, however, a third explanation which I now desire to bring forward, namely, that the infection takes place from the filtration angle. Strong evidence that this is the true explanation is afforded by the changes in the region of the filtration angle in this case. At the sites of the old scleritis the vascularization and scarring of the tissue extended directly from the ligamentum pectinatum (Figs. 6, 7, and 9). That the changes in and about the latter were not due to extension forward of scleritis occurring primarily farther back was shown by the fact that in some places these changes were limited to this region. At the site of the active scleritis (Fig. 8) the inflammatory changes likewise lead directly from the ligament, within which active foci of epithelioid cells were found.[1] Accepting this explanation, it becomes obvious that the infection is carried to the filtration angle by the aqueous humor. As possible sources for the infection of the aqueous humor, the exudates on the ciliary processes in this case suggest themselves. These were too recent, however, to explain the original scleritis. Moreover, no such lesions have been found in other cases reported, and in this case there were no old scars recognizable where similar lesions might have previously existed. On the other hand, it might well be that these exudates were unusual only in regard to size. It is even conceivable that when of such comparatively large size as in this case they might heal without leaving recognizable scars. Still smaller ones could undoubtedly do this. However this may be, the occurrence of these lesions serves to establish the fact that tuberculous metastases may readily lodge in the superficial capillaries of the ciliary body, a fact that, *a priori*, seemed obvious. And it is not necessary to assume that definite lesions should always result for infection of the aqueous humor to ensue, for bacilli might readily pass through the intact pars ciliaris retinæ when carried in leukocytes or endothelial cells. Exudates on the posterior surface of the iris, similar to that in the present case, also

[1] Compare also Komoto's Fig. 4.

offer possible sources for the infection of the aqueous humor, but, no doubt, they play only a subordinate part.

It may be asked here why it is that, if, under these circumstances, lesions usually fail to develop, or undergo retrogression in the ciliary body, metastatic foci should form in the sclera. This can easily be explained by the poor capillary blood-supply of the latter. It must be remembered, too, that the lesions in the sclera, while sometimes of considerable size relative to the eye, are absolutely very small and show decided tendency to heal. Another question which arises is whether the recurrent attacks of scleritis are due to renewed activity of old foci or to fresh metastases. The frequency with which the second eye becomes affected indicates that the latter is often the case.

This explanation of scleritis obviously applies with equal force to the lesions of tuberculous keratitis. Where the latter occurs without definite scleritis, as it rarely does primarily, the infection, no doubt, is directly from the filtration angle, and produces either deeply seated discrete metastatic foci (keratitis punctata profunda) or extensions from the ligamentum pectinatum. Where scleritis has occurred, the infection may, in addition, take place similarly from foci in the sclera, giving rise to more superficial discrete foci, or, by direct extension, producing the so-called sclerosing keratitis.

The character of the adhesions of the root of the iris to the ligamentum pectinatum in this case (Figs. 3, 4, and 8) accords well with this theory. Unlike the adhesions in glaucoma, they do not involve the apex of the filtration angle, and occur only in certain sharply localized spots. The incompleteness of the adhesions is not an artefact, because this was recognized before the eye was placed in alcohol. In most instances the union is firm, evidently old, but one adhesion is found still in the formative stage. The appearances here are almost if not quite conclusive that the adhesion is due to the contact of the iris with an exudate which had formed over the central portion of the ligament. The exudate is obviously to be explained as the result of tuberculous metastases from the aqueous humor.

This theory of the origin of tuberculous scleritis and keratitis gains additional support from the recent important work

of Henderson[1] on the filtration angle. The significance, in this connection, of the following paragraph from one of his papers is apparent (page 172): "The main vessels of the venous system of the iris and ciliary body, including the afferent and efferent veins of Schlemm's canal, are surrounded by perivascular lymph-spaces. In the case of Schlemm's canal the sheath is absent on the inner side, so that the aqueous comes into direct contact with the walls of the sinus. I have never found pigment travelling along in a vessel, but always outside in the perivascular sheath. This is certainly a most important point which one would certainly dwell on if one were discussing the more detailed physiologic instead of the anatomic aspect of the aqueous outflow."

This fact, emphasized by Henderson, that the spaces of Fontana are connected with the perivascular lymph-spaces, explains why the nodules of scleritis occur so often in the region of the anterior perforating vessels, which he has shown elsewhere are almost all connected indirectly with the canal of Schlemm. He has shown also (p. 55) that the spaces are continuous with the corneal spaces, which explains how the cornea may be infected by extension or metastasis from the filtration angle. Henderson has pointed out also (p. 52) that the spaces of Fontana communicate with the suprachoroidal lymph-space. This suggests that the foci in the choroid, which occasionally occur in scleritis, are due to metastases carried from the filtration angle. More likely, however, they are analogous to the foci on the ciliary processes in this case, that is, they are due to blood metastases lodging in the choriocapillaris.

Support is also given to this theory by a case of highly malignant intraocular sarcoma, which I have recently described.[2] In this case a small sarcoma of the ciliary body situated in the upper-outer quadrant gave rise to numerous minute metastases in the filtration angle, and one compara-

[1] "The Anatomy of the So-called Ligamentum Pectinatum Iridis and its Bearing on the Physiology and Pathology of the Eye," *Trans. Ophthal. Soc. of the United Kingdom*, 1908, p. 47; also, "The Venous Connections of Schlemm's Canal," *ibid.*, p. 168.

[2] "A Small Sarcoma of the Ciliary Body Showing Some Unusual Manifestations of Malignancy," *Ophthalmology*, July, 1908.

tively large nodule in the episcleral tissue contiguous with the corneal limbus on the nasal side. The analogy to tuberculous scleritis is here almost perfect.

The occurrence of the metastatic tumor nodule at the inner side, instead of below, as might have been expected, brings up the question as to the most frequent sites of the foci of scleritis. To obtain, if possible, definite information on this point, Dr. Wm. Liebman, at my suggestion, has kindly examined the house records of the infirmary and tabulated the positions of the lesions recorded in a consecutive series of cases of scleritis and sclero-keratitis.[1] Both recent nodules and old areas of bluish discoloration were counted. Out of 46 lesions (42 cases), they occurred below only 3 times; above, 24 times; to the nasal side; 12 times; to the temporal side, 7 times. They occurred in the upper-outer quadrant 15 times; in the upper-inner quadrant, 5 times. These statistics show conclusively that the lesions are much more frequent above than below. On the theory of scleritis that I have just advanced this is difficult to explain, but it is equally so on any theory. The frequent occurrence of lesions to the inner and outer sides is explainable as due to the position of the eye with reference to gravity during sleep. A possible explanation of their still more frequent occurrence above is that it is in some way dependent upon the repeated traumatism to the upper part of the sclera incident to the act of winking. This almost constant irritation might well cause small metastatic foci to reach macroscopic size, which elsewhere would undergo retrogression. That even slight trauma may stir into activity quiescent lesions I have several times had occasion to observe in the experimentally produced tuberculosis of animals.

Conclusions.

In tuberculous scleritis (anterior nodular scleritis), the infection reaches the sclera through the filtration angle.

[1] Statistics as to the situations of the deep lesions of tuberculous keratitis would also be desirable, but at present are not obtainable, owing to the fact that clinical notes are not made with this object in view. Judging, however, from a few recent cases, these lesions are most common below.

The infecting bacilli are derived chiefly from the superficial vessels of the ciliary processes, and are carried in the aqueous humor to the filtration angle. Whether there are always small initial foci on the surface of the ciliary processes, or whether the bacilli, contained probably in phagocytic cells, are exuded through the intact pars ciliaris retinæ, is uncertain.

In tuberculous keratitis the infection is also from the aqueous humor, the cornea becoming infected either by metastasis or extension from foci about the filtration angle, or in a similar manner from the foci of a preceding scleritis. In pure tuberculous keratitis, which rarely, if ever, occurs, the infection takes place in the former way only.

Two forms of tuberculous cyclitis are to be distinguished, namely, the interstitial and the superficial (punctate). Interstitial cyclitis is usually secondary to scleritis, and due to the direct extension of the latter. Superficial cyclitis occurs as direct blood metastases. Clinically, it manifests itself as one of the forms of serous cyclitis.

Superficial tuberculous iritis, involving the posterior surface of the iris, may occur, and is analogous to superficial cyclitis, with which it is probably often associated.

To Illustrate Dr. Arnold Knapp's Article on "Glaucoma from Adhesion of Lens Capsule to the Cornea."

Fig. 1

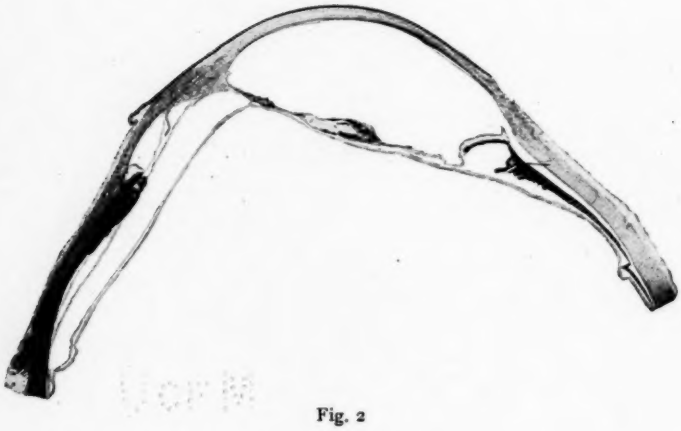

Fig. 2

GLAUCOMA FROM ADHESION OF THE LENS CAPSULE TO THE CORNEA.[1]

By Dr. ARNOLD KNAPP.

(With two illustrations on Text-Plate XV.)

THE lens capsule is apt to become adherent to the cornea after a penetrating injury to the eyeball, where the cornea and lens have been wounded, or after the extraction of cataract. In some cases this is followed by glaucoma which is very difficult to control and is often followed by blindness. In the first or traumatic variety I should like to report on three cases.

I. Traumatic Irideremia. Broad Adhesion of Capsule to Transverse Corneal Wound. Glaucoma.

D. G., 24 years old, while riding in an open surface car received a severe blow in the left eye, one week before her first visit on August 12, 1904. The eye was congested, there was a transverse wound across the centre of the cornea, the anterior chamber contained cataractous lens material, and no iris was to be seen. Tension normal. Field normal. Vision: m. h. After two months, October 8th, the eye was quiet, the lens completely absorbed, the capsule was broadly adherent to the corneal scar. Tension +. Nov. 7th. The capsular adhesion was divided with the pince-ciseau+. No reaction. Vision with correcting glass c = $\frac{8}{10}$. Jan. 14, 1905. The inferior capsule had again become united to the corneal scar. Tension normal. April 5th, 1905. Vision = $\frac{8}{10}$. The upper opening has become partly filled in with new tissue. Tension +½. April 23, 1906. The operation was repeated as the tension was increased. A round opening remained in the capsule. Tension

[1] Read at the meeting of the American Ophthalmological Society, Washington, May, 1910.

normal. Is using pilocarpin drops. Vision = $\frac{20}{40}$. October 12, 1907. Tension has been again elevated for some time. At another operation it was attempted to resect a V-shaped piece of capsule; this failed. The tension again became raised. It has been kept somewhat in check by miotics though now it is slightly raised and vision is only $\frac{20}{40}$. Field and fundus are normal.

II. Traumatic Irideremia. Broad Adhesion of Capsule to Cornea. Glaucoma. Enucleation. Pathological Examination.

A. C., 30 years old. After a severe blow to the right eye on December 2, 1907, the eye three weeks later presented a transverse scar in the centre of the cornea with absence of iris, absorption of lens, and a thickened capsule adherent to the corneal scar. The tension is increased. On Jan. 20, 1908, the glaucoma has been severe and the eye painful; cyclodialysis was performed as from the experience in the previous case division of the capsular adhesion was not promising. A quadrant of the ciliary body was detached. This was followed by hemorrhage into the anterior chamber with no reduction in tension and continuance of pain. Pilocarpin sweats and miotics were of no avail. Enucleation was refused, and the patient disappeared from view until January, 1910. On her return she stated that the eye had quieted down until quite recently; now it was blind, inflamed, and painful. The eye was excised and prepared for ***microscopic examination***. (Fig. 1.)

This showed that there was no iris. The ciliary processes were drawn forward anterior to the pectinate ligament and flattened out, occluding the angle of the anterior chamber. The apices of the ciliary processes were distinctly to be seen drawn forward by the zonular fibres in the direction of the corneal scar. Both capsules are adherent to the corneal scar. There is narrow space between the capsular layers filled with degenerated lens fibres, vesicular cells, lenticular débris, and organized connective tissue. The place of adhesion to the cornea is a somewhat thickened area comprising cicatricial tissue and convoluted Descemet's membrane.

III. Broad Adhesion of Capsule to Cornea. Glaucoma.

A boy aged 12 years, on being examined for a refractive condition was found to be blind in one eye. There was a broad adhesion of iris and capsule to the cornea, no lens,

the eye was hard and somewhat enlarged. There was an indefinite history of an injury several years ago.

After cataract extraction I have observed three cases of glaucoma where the lens capsule was adherent to the corneal incision.

1. COMBINED EXTRACTION. ANTERIOR CAPSULAR ADHESION. GLAUCOMA. ENUCLEATION. PATHOLOGICAL EXAMINATION.

D., 56 years old. Opacities of cornea from interstitial keratitis, posterior cortical cataract. After an extraction with iridectomy, the eye developed glaucoma and the capsule was observed adherent to the corneal section which was rather more corneal than usual. Repeated division of this adhesion was performed without lasting effect; the regeneration of the capsule started from the angles and the interval was soon bridged over again, and glaucoma returned. The eye was finally enucleated on account of the constant discomfort.

Microscopic examination. (Fig. 2.) At site of iridectomy the iris stump is bulbous and turned back(entropion uveæ); the ciliary processes on this side are flattened and pushed forward to the anterior margin of the iris stump. Both layers of the capsule are drawn to and adherent to the corneal scar where the corneal stroma is thickened with scar tissue and convoluted Descemet's membrane. On the opposite side, the side away from the corneal section, the ciliary processes are drawn directly inward in the direction of the scar. The ciliary body is retracted, the iris is atrophied at its root and broadly adherent to the cornea effacing the angle; the free end of the iris is directed inward.

2. COMBINED EXTRACTION. ANTERIOR CAPSULAR ADHESION. GLAUCOMA. CYCLODIALYSIS.

B., 58 years old. Extraction of cataract with iridectomy in a very small, hypermetropic eye. The healing was delayed, the anterior chamber remained unformed for some days. Subsequently the eye showed a tendency to increased tension. There was an anterior adhesion of the lens capsule to the section, which was rather corneal. Glaucoma developed which was not influenced by miotics. Broad division of the capsular adhesion failed to have any

lasting effect. Cyclodialysis was performed in November, 1909. This has kept the tension down to the present date (April, 1910) though there is some diminution in vision.

3. COMBINED EXTRACTION. GLAUCOMA FROM CAPSULAR ADHESION. CURED BY OPERATION.

A. G., 75 years old. Extraction with iridectomy. Normal healing and good visual result. Two months later the eye was found to be hard. On careful examination a narrow band of capsule at outer column of iris coloboma was found to be attached to the corneal incision. This was divided with a knife needle and the tension became normal. One month later the eye was again hard and the band of capsular adhesion had re-formed. The strand was then drawn out with a Tyrrel's hook and divided, the adjoining capsule was cut with pince-ciseaux, causing slight prolapse of vitreous. The band has not re-formed, the tension is normal, and vision undisturbed.

The cause for the glaucoma seems to be the obliteration of the angle of anterior chamber by the ciliary processes or iris displaced forward by traction of the capsule adherent to the corneal scar as Treacher Collins (Trans. Ophthal. Soc., United Kingdom, 1890, *Royal London Ophthalmic Reports*, vol. xvi., part iii.) has shown. This at least is the condition found on microscopic examination. The mere mechanical obstruction of the angle of the anterior chamber cannot be the only or the primary cause, as the cases, where only a small part of the capsule is adherent, show.

The traction is greater the more central the corneal scar and the glaucoma is proportionately more severe. The total absence of the iris in the first two cases had no effect in counteracting the glaucomatous process. It also shows the uselessness of an iridectomy as a remedial agent. When the scar is central and occupies the greater part of the corneal diameter we are powerless to prevent the progress of the glaucoma. Operations to divide the capsular adhesions seem even to make matters worse, as the regeneration is so active, and the resulting capsular adhesion is denser than before.

In the cases following cataract operation, preventive treatment is most important. This condition occurs when

the extraction has been performed with iridectomy and is facilitated by certain methods of opening the capsule. The avoidance of capsular complications is the greatest advantage of the simple operation. The importance of recognizing the dangers of capsular adhesions is accepted by all operators and it is common practice to stroke back any capsular tags in the region of the iris coloboma. This cannot be done with certainty as the capsule is invisible. Delayed union with unrestored anterior chamber naturally predispose to this accident. The treatment after the condition has occurred and the eye is hard, is to divide the adhesion. If the adhesion is broad it is difficult and unsatisfactory; when the adherent band is narrow it can be readily divided in a manner to prevent its reuniting and the glaucoma is cured.

ON OPTIC-NERVE DISEASES DUE TO POISONING WITH ORGANIC AND INORGANIC ARSENICAL PREPARATIONS.[1]

By Dr. OTTO SCHIRMER, New York,

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(*With 4 figures in the text.*)

ARSENIC has been freely used in medicine for more than a century. Internal medicine and dermatology especially have taken advantage of its tonic qualities and its curative effect on many skin and blood diseases, the etiology of which is not yet known. Experiences of recent years have shown that the organic arsenical preparations are more efficacious than the inorganic salts.

Atoxylon, the first organic compound of arsenic, was used in 1902 for the treatment of skin diseases. It was tried first by Schild.[2] It soon became well known by the reports of Robert Koch, who used it extensively and with good results in the treatment of sleeping-sickness in Africa. Following these reports it was soon tried in Europe in the treatment of a great many diseases caused by protozoa, such as syphilis, malaria, recurrent fever, and the like. After some time, however, there were reported some untoward effects of the drug, the most important of them being blindness. Koch[3] reported 23 cases of this complication out of 1633 patients treated with atoxylon. Ophthalmoscopic

[1] Read before the Ophthalm. Section, N. Y. Acad. Med., April 18, 1910.

[2] *Berlin klin. Wochenschrift*, 1907, p. 1889.

[3] *Deutsche medic. Wochenschrift*, 1902, p. 279.

examination showed later that the blindness was caused by an optic atrophy. It was, however, doubtful whether the latter was a consequence of the treatment or a complication of the disease. In the following years other similar cases were published in which the primary disease could not have been the cause of the blindness. Their number is relatively small; I did not find more than 25 cases in the literature—in addition to the 23 of Koch. There is no case published in America or England.

For this reason I take the liberty of reporting to you the following case which came under my observation in Strassburg. It was a typical case of atoxylon-poisoning in which the French preparation was used which is believed to be purer and less dangerous than the German preparation. It was as far as I knew the first optic atrophy after the French atoxylon; later Coppez observed a similar case.

CASE I.—Male, 58 years old, was sent to me on the 17th of July, 1908, from the Skin Hospital because he complained of dimness of vision and scintillation.

For 26 years he had been suffering from a mycosis fungoides which was treated 6 years ago with sodium arsenite and again in September and October 1907, when he received 30 injections. They were stopped on account of symptoms of general intoxication, such as tremor, excitement, anorexia, and paresis of the right leg. After a good recovery from these symptoms, the arsenic was given again in December, but stopped after a few weeks, on account of general malaise and diarrhœa. The total doses of arsenic given in inorganic preparations amounted to 3.6 grammes.

The first of May, 1908, he received injections of atoxylon. It was given in increasing doses of 0.1 gramme to 0.8 gramme and then reduced again to 0.1 gramme. He received 52 injections, two every week, the total amount of atoxylon given being 9.9 grammes. At the end of May the patient began to suffer from scintillation. Distinct diminution of vision, however, did not appear before the beginning of July.

When I first examined the patient I found a sluggish and diminished reaction of both pupils, their diameter being very large. I also noticed fine opacities in the vitreous body. Both disks were very pale, sharply outlined, the arteries contracted. The vision was $\frac{2}{10}$ in the

right and $\frac{1}{10}$ in the left eye. The visual field was much contracted especially to the nasal side. (See Fig. 1.) No color scotoma; patient is an abnormal trichromate. No general symptoms of intoxication except increased knee-jerks. Urine free from sugar and albumen, the Marsh test for arsenic being positive.

The treatment consisted in the administration of iodide and bromide of potassium and strychnia, and he was ad-

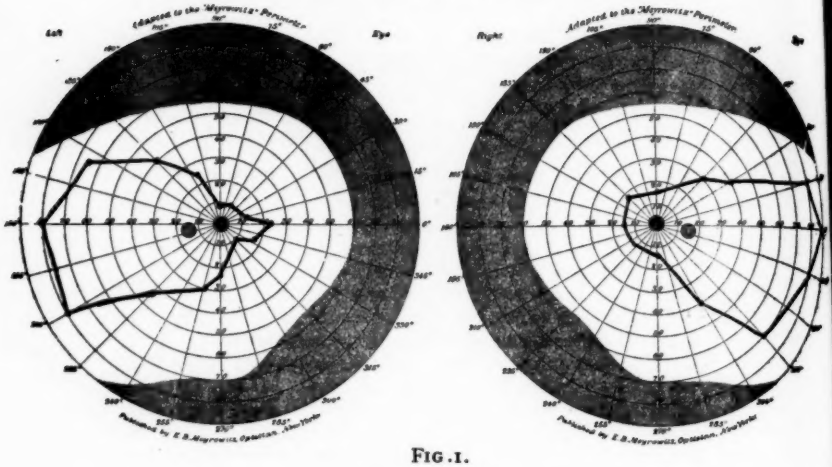


FIG. 1.

vised to drink large quantities of fluid in order to increase diuresis.

August 12th. Disks paler than before, arteries and veins very much narrower, vision $\frac{1}{10}$ on the right, $\frac{5}{10}$ on the left side. Field much more contracted. (See Fig. 2.)

September 10th. Arteries and veins very narrow. Vision of right eye unchanged, of the left only light perception.

October 9th. R. E. V. $= \frac{5}{10}$; left eye quite blind.

November 13th. R. E. V. $= \frac{3}{10}$; visual field very narrow but colors recognized in centre.

January 29th. Only light perception in the right eye. Both disks grayish white, without the slightest trace of red, arteries and veins extremely narrow.

When I saw the patient last during the summer of 1909 the right eye also had become absolutely blind, his general condition during this whole period being fairly good, without any signs of general intoxication.

As soon as the value of atoxylon was recognized, it was only natural to try and find a similar organic compound which embodied the curative properties of the drug without its toxic after-effects. Ehrlich especially worked along these lines and the first preparation he brought forward was arsacetin. Atoxylon is the monosodium salt of paramido-phenyl-arsenic-acid. Arsacetin contains instead of one hydrogen

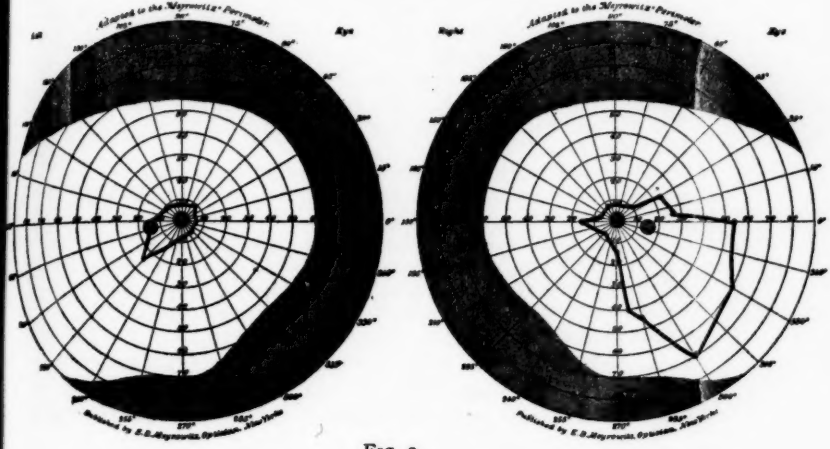


FIG. 2.

atom of the amidogroup an acetylgroup and represents therefore the monosodium salt of the paracetyl-amido-phenyl-arsenic-acid. It is according to Ehrlich's experiments only $\frac{1}{10}$–$\frac{1}{4}$ as poisonous as atoxylon for mice, and destroys trypanosomata in the same concentration as the former.

Arsacetin has been used in the treatment of a large number of cases of syphilis by Neisser and Heymann; and for pernicious anæmia by Klemperer. I had occasion to observe the first case of optic atrophy due to arsacetin; it was sent to me from the Skin Hospital in Strassburg. It has been reported already by Dr. Ruete,[1] a dermatologist. At that time the optic atrophy was still in its beginning and I therefore shall report the case in extenso.

CASE 2.—Male of 73 years suffering for years from a

[1] *Münchener medic. Wochenschrift*, 1909, p. 718.

very stubborn psoriasis for which he received 48 injections of sodium arsenite in 1906 and 18 injections in 1907. Slight arteriosclerosis. He received the first injection of 0.6 gramme of arsacetin on the 3d of February and 5 injections more during that month. Then it was stopped because the patient complained of scintillation, foggy vision, and indistinctness of peripheric vision, the total amount of arsacetin given being 3.6 grammes.

I saw him first on the first of March. The pupils were of normal size and reacted well. In the vitreous body there were fine opacities; both disks were pale, sharply outlined, and both sets of vessels narrow. The vision was still $\frac{1}{3}$ on the right, $\frac{1}{4}$ on the left side. The visual field was very narrow, especially nasally and principally on the left side. The treatment consisted of iodide of potassium, strychnia, and electricity.

Five days later the vision had already decreased to $\frac{1}{4}$ in the right and $\frac{1}{10}$ in the left eye. The visual field was still more contracted, the disks still paler.

On the 20th of March the left eye was quite blind, the vision of the right unchanged. This condition remained the same until the end of May. Then the right also began to fail more and more and the blindness was complete at the beginning of July.

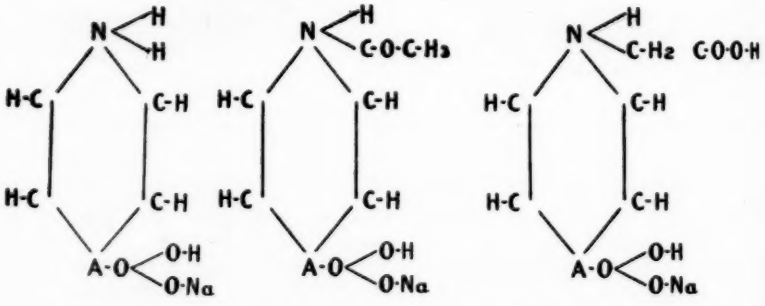


FIG. 3.

It is not necessary to point out the similar features in both of my cases. In five other cases reported in the recent months by Iversen,[1] Hammer,[2] and Eckhardt[3] we find the same

[1] *Münchener medic. Wochenschrift*, 1909, p. 35.

[2] *Deutsche medic. Wochenschrift*, 1910.

[3] *Archiv für Schiffs- und Truppenhygiene*, vol. 13., H. 16 ,1909.

ophthalmoscopic changes and the same disastrous outcome.

Still other organic arsenic compounds, very similar in their construction to atoxylon and arsacetin, have been devised by Ehrlich and his pupils and have been proved to have a specific action on trypanosomata both in animals and human beings. Wendelstadt[1] reported in 1908 experiments with three new arsenical preparations on rats infected with trypanosomata. He found them able to destroy the protozoa and in some instances to save the life of the rats, but a couple of them became blind. This accident was observed twice after treatment with paroxy-benzyliden-arsanilacid and several times after arseno-phenyl-glycin. But this drug proved to be noxious only when preserved in the usual way; when preserved in a vacuum it did not produce blindness, and Wendelstadt suggested therefore that the unfavorable effect was due to a disintegrated drug, it having been changed into arsen-oxyphenyl-glycin. This blindness of the rats was due to an optic atrophy; their disks, reddish under normal conditions, were found to be gray and the vessels were extremely narrow. It was exactly the same picture as observed in rats after atoxylon and arsacetin administration.

Some weeks ago there was published by Alt[2] a report on a series of injections made in 140 paralytics with positive Wassermann reaction. In no one case was there impairment of vision, but Alt has treated all these patients with two injections only, each of one gramme given two days in succession. There is still another favorable report on arseno-phenyl-glycin in the literature published by Eckhardt,[3] who treated nineteen patients with sleeping-sickness but also gave only four grammes in six injections. I do not think it is justifiable to conclude from these experiences that the arseno-phenylglycin does not act at all on the optic nerve. The number of patients is too small and I may remark here that in the first reports on arsacetin given by Neisser, Heymann, and Klemperer there was no case of blindness.

When we survey all the cases of blindness observed after treatment with these organic arsenical compounds there can

[1] *Berliner klin. Wochenschrift*, 1908, Nr. 51.
[2] *Münchener medic. Wochenschrift*, 1910, p. 561.
[3] *Loc. cit.*

be no question that this accident is due to their toxic action on the optic nerve. The atrophy of the optic nerve does not depend on the primary disease as was supposed at first and still is claimed by some dermatologists. This supposition cannot be upheld any longer for two reasons:

1. Blindness occurred in several diseases never known to produce optic atrophy. It occurred even in rats which were infected with trypanosomata and treated with arsenic compounds, the infection with trypanosomata alone never having produced optic atrophy.

2. There exists quite a typical clinical feature of optic atrophy due to compounds of the atoxylon group which is distinctly different from the optic atrophy due to syphilis and to other intoxications. Only quinin produces a similar picture, but the course is different, as partial recovery is the rule.

The characteristic features are exactly the same in all organic arsenical compounds of the atoxylon group. It seems therefore that the primary drug is not the poisonous one, but that it is changed in the body by reduction into other compounds which are perhaps the same in all these different preparations. Ehrlich first made this suggestion in reference to atoxylon, having found that it is not able to destroy the trypanosomata in the test-tube but only in the living body. He succeeded by reducing the atoxylon in obtaining two very poisonous substances: paramino-phenyl-arsenoxyd and diamido-arseno-benzol. Each of them destroyed trypanosomata in the test-tube in a few minutes. Contrary to Ehrlich, Igersheimer[1] states that the atoxylon leaves the body unchanged—except for small quantities, which are changed into inorganic arsenic, but he admits that it is perhaps transformed only in the cells.

The characteristic and common features of this optic atrophy are: onset with haziness, scintillation, and contraction of the visual field, especially on the nasal side, the same as is found in quinin-amblyopia. The central vision remains fairly good until the field has become very narrow. The optic nerve becomes pale with sharp boundaries in the ma-

[1] *Archiv für Ophthalmologie*, Band 71, p. 379, 1909.

jority of observations at an early date. In Koch's cases the discoloration was found later. There is contraction of the arteries at the onset and, in the late stages, when the disk has got quite white, both sets of vessels are extremely narrow. As a rule, the atrophy runs a slow but progressive course. It takes months before the eye becomes entirely blind and this is avoided only in exceptional cases. I have no doubt that some patients have eventually become quite blind, who, when last seen, had still some sight left. In some cases temporary arrest of the process has been noted. Only in two cases of Fehr[1] there seems to have been a favorable outcome. He observed two patients who, two years after the beginning of the atrophy, had normal vision; the visual field was contracted, especially on the nasal side, and the condition of the disk was quite characteristic. I cannot agree, therefore, with Igersheimer that this atrophy is due to small quantities of inorganic arsenic set free from the atoxylon in the body. Barring the favorable outcome, the clinical features in these two cases are exactly those of atoxylon-atrophy and quite different from the inorganic arsenical optic-nerve affections. The seven cases of Koch mentioned later by Beck[2] in which there was only slight impairment of vision cannot be made use of, because they were not observed closely enough.

The question whether this unfortunate accident can be avoided with certainty must be answered in the negative. The smaller the dose, of course, the slighter the danger, but there is no doubt that the chief point is the individual disposition. While most patients stand rather high doses without any ill effects, there are persons whose optic nerves react unfavorably even to small quantities. While many persons have borne well 20 to 30 and even more grammes of atoxylon and 15 to 20 grammes of arsacetin, the patient of Hammer[3] became blind after 8 doses of only 0.1 gramme of arsacetin. Iversen's[3] patient received only 0.7 gramme and five days later 0.5 gramme arsacetin. The favorable cases of Fehr stood even a total amount of about 25 grammes of atoxylon.

[1] *Deutsche medic. Wochenschrift*, 1907, Nr. 49.
[2] *Centralblatt für prakt. Augenheilkunde*, 1909, p. 129.
[3] *Loc. cit.*

It is very uncommon that two doses following each other at such short intervals are sufficient to produce the blindness. As a rule, smaller doses continued for a longer time, especially repeated injections, are more dangerous and there are often found in the history previous injections of inorganic arsenical preparations. The previous use of mercury and potassium seems also to increase the danger. For the prevention of blindness it is therefore much safer to give only one large dose of the preparation, Ehrlich's *dosis magna sterilisans*, instead of long or repeated weak injections. Discontinuing the injections as soon as the first eye-trouble is complained of is of no avail; for the atrophy will continue until the entire nerve is degenerated.

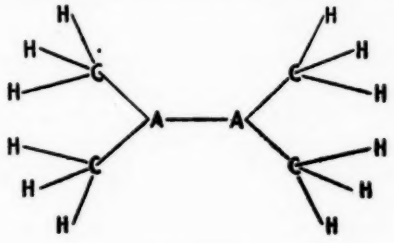
Since arsenic is extensively used as a drug, it is an important question whether these bad results reported after the use of atoxylon depend upon the arsenic molecule or are inherent properties of the atoxylon group. In searching the literature for optic-nerve affections due to inorganic preparations, I could collect only a very small number. This is the more remarkable when we remember how extensively arsenic has been used for an entire century. We know that eating arsenic is a frequent custom in some countries and is continued for years and years without severe eye-symptoms. It is worth mentioning that in 1900 in Manchester 1000 persons were badly poisoned with beer containing arsenic. In many of these cases conjunctivitis and œdema of the lids were observed, but not one case of optic atrophy, although all the patients were examined carefully.

I did not succeed in finding more than six cases[1] of optic-nerve affections after inorganic preparations and no one showed the same features as the atoxylon-amblyopia. Here we have to deal with a primary atrophy as can be concluded from its clinical appearance and as is proved by experimental researches in rabbits, dogs, and cats by Igersheimer. The

[1] Dana, *Brain*, vol. ix., p. 456, 1897; Derby, *Boston Med. and Surg. Jour.*, vol. cxxiv., p. 602, 1891; Liebrecht, *Klin. Monatsbl. für Augenheilk.*, vol. xxix., p. 181, 1891; Meirowitz, *Journal of Nervous and Mental Diseases*, vol. xx., p. 170, 1895; Brouardel, *Arch. de méd. expér. et d'anatom. path.*, Nov., 1896; Lanceraux, *Gazette des hôpitaux*, 1896.

only post-mortem examination in man by Nonne[1] showed also in the optic nerves simple atrophy of the fibres without inflammatory changes in the interstitial tissue. There has been no post-mortem examination of inorganic arsenic poisoning and the experimental researches of Igersheimer do not reveal any changes in the nerve tissue of the eyeball except slight alterations in the ganglion cells in the retina; but the clinical features point to an inflammation in the nerve tissue.

These clinical features are the following: There is always a moderate impairment of vision only, the visual field being normal, but a central scotoma for colors being present. The disk is not quite sharply outlined, the arteries are narrow, the



KAKODYL

FIG. 4.

veins unaltered in size, later there appears pallor of the temporal half. The prognosis is a very good one; recovery occurred in all cases after discontinuing the drug. Not one case of total blindness is reported. On the other hand the inorganic arsenic is especially liable to produce conjunctivitis and œdema of the lids, a condition never met with in atoxylon poisoning.

Another organic arsenical compound used in medicine is kakodyl and its derivations. Kakodyl is tetra-methyl-arsen. Although it has been used more freely in recent years I did not succeed in finding any cases of optic-nerve affections.

In concluding I want to emphasize that there exists an essential difference in the pharmacodynamic action between

[1] *Medicinische Klinik*, 1908, Nr. 20.

organic and inorganic arsenical preparations. The latter damage the optic nerve only in exceptional cases, producing a retrobulbar optic neuritis of about the type of the ethyl-alcohol-neuritis, both of which give a favorable prognosis. Progressive optic atrophy, in contradistinction to the last-mentioned retrobulbar neuritis, originating without inflammatory changes and ending in blindness, is characteristic of the organic arsenical preparations of the atoxylon group. Kakodyl is as far as we know not liable to produce it.

IMPLANTATION OF A METAL BALL IN TENON'S CAPSULE (THE FROST-LANG OPERATION), WITH REPORT OF 48 CASES.[1]

By Dr. WILLIAM M. SWEET, Philadelphia.

DURING the past six years I have performed the Frost-Lang operation of implanting a ball in Tenon's capsule in every instance in which removal of an eyeball was required, when no contra-indication existed. The total number of cases was 48, with loss of the ball in 2 cases.

The reasons for the removal of the eyeball may be thus classified:

Twenty-two cases of injuries from foreign bodies: in 6 extraction failed, and the eye was at once removed; in 5 extraction was successful, but blindness with shrinkage of the globe ensued; and in 11 extraction was successful, but immediate or remote iridocyclitis developed.

Ten cases of punctured or incised wounds of the eyeball by sharp instruments or large pieces of glass or other materials.

Two cases of ruptured eyeball from blows.

Four cases of blindness with painful glaucoma, in one of which, a woman aged forty-nine years, a small choroidal sarcoma was found.

Four cases of corneal staphyloma, 2 in adults following gonorrhœal infection, and 2 in babies, one eight months and the other twenty months old.

Four cases of dense corneal leukoma, the enucleation being performed for cosmetic purposes.

Two cases of necrosis of the cornea, 1 a man aged fifty, and the other a woman aged fifty-eight.

[1] Read at the meeting of the American Ophthalmological Society, Washington, May, 1910.

In 5 of the cases of injury there was evidence of sympathetic irritation at the time of the enucleation, with the injured eye tender upon pressure, and in 2 cases considerable photophobia. In every instance there was subsidence of the symptoms following the enucleation, and no reaction occurred from the insertion of the gold ball.

In 42 cases gold balls were implanted, the ball varying in size from 13 to 15*mm*. The 14*mm* ball was the size most often adapted for adults. In 6 instances platinum instead of gold balls of the above sizes were substituted.

In the 2 cases in which the ball was lost an excellent stump was obtained, with considerable movement of the artificial shell.

The recognized advantages of the operation of implantation over that of simple enucleation are:

First: The stump in practically every case gives a more movable artificial eye than ordinarily follows simple enucleation. In some cases of enucleation in which the muscles have been sutured to the conjunctiva and care exercised to preserve all conjunctival tissue, an excellent movable prosthesis is secured, but the operator is not certain that this result will be obtained.

Second: The depression of the tissues immediately beneath the brow and the tendency to enophthalmos of the artificial eye are less marked.

Third: The floor of the orbit after healing is flat, with occasionally a slightly raised portion in the centre where the implanted ball projects forward, a condition superior to the deep, furrowed, and often irregular socket for the retention of secretion that so often follows simple enucleation.

With the exception of eyes with malignant growths and those purulently inflamed, there is no contra-indication to the operation of implantation. I do not feel, however, that the insertion of a gold ball after the removal of an atrophic stump is a satisfactory procedure, because all the tissues are shrunken and even a small sphere must exert more pressure upon the orbital tissues than is the case after the removal of a full-sized eyeball. In the latter the capsule contracts and forms a firm envelope around the sphere, and the nerves are retracted beyond the reach of dangerous pressure. In the

severe types of traumatic iridocyclitis requiring enucleation, even though the other eye has become ifritated, there is, in my opinion, no greater danger of sympathetic ophthalmitis than if the ordinary enucleation was performed without a gold-ball implantation. I believe that should sympathetic ophthalmitis appear it would have occurred no matter what operative procedure had been followed.

In 6 of the recent cases platinum balls, and in the other 42 gold balls, were implanted. Although it does not seem possible that the gold ball can be affected by the secretions of the orbit, I feared that they might become so, therefore a number of platinum balls were made with the intention of using this metal in future operations. As yet, however, I have not been able to secure balls of platinum as light in weight as those of gold. An X-ray plate of a case in which the gold ball was implanted six years ago shows the sphere intact and free from erosion. Most of the earlier gold balls used were composed of two half spheres soldered together, and the work was so perfectly done that it was impossible to see the point of joining. Recently the makers have cast the full ball around a central core, and the latter is removed through a small opening which is subsequently closed. In both instances the high heat to which the metal is subjected insures perfect sterility of the interior of the ball.

The method of operation followed does not differ in any important particulars from the usual methods that have been described.

After the conjunctiva has been dissected loose at the limbus, and the usual precautions to preserve all the structure have been observed, the four straight muscles are picked up on the hook and stitched to the conjunctiva in the normal position or brought forward to the edges of the cut conjunctiva. After the nerve has been separated and the eyeball removed, hemorrhage is stopped by pressure of dry sterile gauze pads, and the upper and lower border of Tenon's capsule picked up with forceps, and the ball dropped into the cavity exposed. A stitch is inserted through the upper and lower edges of the centre of the capsule and tied, one end of the thread cut off while the other is used to raise the capsule, in order that the sutures may be more readily inserted. From

6 to 8 will usually be required to join the edges of the capsule. Should any portion of the capsule be so thin that the ball shows through, a tuck of the adjoining tissues should be made and the thinned portion covered. The conjunctival edges are now brought together by interrupted sutures placed horizontally. A binocular bandage is applied, but no iced compresses are employed. At the dressing the following day the monocular bandage is used, and the patient allowed to get up. Should œdema of the lids be present, cold formalin applications may be made through the light bandage.

The complications of the operation of implantation are claimed to be: (1) Swelling and ecchymosis of the lids; (2) escape of the ball; (3) displacement of the ball after healing; (4) sympathetic trouble.

(1) *Protracted Convalescence.*—The reaction following the operation of implantation is usually no greater than after simple enucleation. There is somewhat more œdema of the conjunctiva, but this is to be expected, since the gold ball occupies a position in the orbit that would be taken up in the simple enucleation by the swollen conjunctiva. In the majority of instances the patients will have left the hospital within a week of the operation. In 5 cases of the series there was œdema of the conjunctiva and swelling of the lids, but this was plainly the result of effusion of blood into the orbital tissues. In these cases there was some difficulty in cutting the optic nerve, so that the blood from the severed orbital vessels probably percolated into the tissues before egress was permitted by removal of the eyeball. In none of these cases, however, was the gold ball lost, although the convalescence was protracted.

(2) *Escape of the Ball.*—In four of the earlier cases the central portion of the wound failed to unite, and the edges separated, exposing the ball. In the first, the exposed portion of the ball measured 8*mm* in diameter, and was first noticed on the sixth day. Believing that the ball would ultimately be lost, and in order to shorten the period of healing, an attempt was made to remove the ball by means of a hook, but the band of tissue surrounding the ball held so firmly and the patient complained so much of the pain that the attempt was abandoned. The edges of the wound were

then touched with the mitigated silver stick, and this was repeated every second day for a week, at the end of which time the conjunctiva and capsule had granulated and completely covered in the ball. In the second case of separation of two of the central sutures, a like procedure was followed by the same gratifying result. Restitching of the conjunctiva over the exposed ball is useless, as the sutures cut out. Lang, in a case of threatened escape of the sphere, removed it, inserted one of smaller size, and brought the wound-edges together successfully.

In the two cases in which the ball was lost one was due to infection of the entire wound-edges and the sphere escaped on the third day. In the second the three central stitches cut out, and although attempts were made to stimulate granulations along the edges of the wound, the tissues would not cover in the ball, and it was removed on the nineteenth day.

(3) *Displacement of the Ball.*—In none of the cases has the ball shown an appreciable change in position. This complication is not so likely to occur in implantation at the time of enucleation as in the operation of remote implantation, since the shrinkage of the capsule forms a firm envelope about the ball and holds it in the central position.

The tying of the severed ends of the four recti muscles in a bunch over the ball, as recommended by some operators, would appear to favor displacement of the ball by the subsequent contraction of the tissues and the forcing of the ball obliquely out of the muscle cone.

(4) *Sympathetic Disease.*—The most important consideration in the substitution of a method for the simple enucleation is the possibility that the introduction of a foreign substance in the scleral cavity, or in Tenon's capsule after the complete removal of the eyeball, may place the good eye in danger. In a majority of reported cases of sympathetic trouble following one of the substitute procedures for ordinary enucleation the operation was done upon injured eyes which, at the time, were the seat of an acute inflammation, and it is impossible to know whether the sympathetic disease would not have arisen irrespective of the nature of operative procedure. The published reports of sympathetic disease following Mules's operation would indicate that this opera-

tion is not without risk, and the same may be said to a less degree of evisceration in which the sclera remains but no sphere is inserted. In the operation of implantation of a sphere in Tenon's capsule the entire eyeball is removed, and presumably all the structures concerned in the causation of sympathetic disease are absent.

Notwithstanding the several instances of sympathetic trouble in cases in which the operation of implantation of a ball in Tenon's capsule was performed, I do not think that in a single instance we can positively point to the implanted ball as the primary and exciting cause of the symptoms in the good eye. So far as I have been able to find, 5 cases of this character have been reported. The detailed history of these cases is as follows:

CASE I.—Reported by Cant.[1] Man, aged thirty, injured in outer ciliary region by a chip of steel. Extraction attempted ten days later, but failed. Patient refused enucleation until a month after injury, then globe removed and a glass ball inserted in Tenon's capsule. Other eye strong and healthy. Three days after operation the glass ball came out, owing to slight suppuration. The same day the good eye was weak and watery, and the next day slight iritis was manifested. This was followed by typical sympathetic ophthalmitis.

CASE II.—Reported by Lang.[2] Girl, aged seven, entered Moorfield's with right eyeball much shrunken, and had been so since birth. There were general pinkish injection, complete posterior synechia, atrophy of iris, and the lens opaque and shrunken. T. n. No. l. p. L. E., healthy. Excision and implantation of a glass ball in Tenon's capsule the day after admission to hospital. Pathologic examination of removed eyeball revealed considerable infiltration throughout uveal tract, increase of cells in optic nerve, and some inflammatory tissue in its sheath. No reaction after operation, and patient discharged eleven days later. Returned eight weeks after operation with iritis of L. E., total posterior synechia, *no keratitis punctata*. T.+2. Under treatment eye improved, but pupil excluded and occluded. In three years linear extraction. Examination

[1] Report of Committee on Excision, Oph. Soc., United Kingd., 1897.
[2] *Ibid.*

six years later, V = fingers at 4 feet. J. 1 at 9 inches. T. n. R. E., socket healthy. Implanted ball displaced up and out.

In a consideration of the possibility of the implanted ball being responsible for the sympathetic disease in the above two cases, the English committee says: "In the first case the interval between the operation and the onset of the inflammation in the sympathizing eye, three days, is so short that we do not think it had anything to do with the onset of the mischief."

In reference to the second case, the committee says: "As the inflammation in the excised eye was apparently spontaneous and not the result of a perforating injury, that of the second eye may have been of a like nature." That the implanted ball was not regarded as the cause of the inflammation in the left eye would seem to be borne out by the fact that the glass ball was not removed, the history showing that it was still in the right socket, although displaced up and out at the examination six years later. I have given the history of these two cases in detail, as they have been quoted for years past as evidences of the danger of the implantation operation.

CASE III.—Reported by Suker. Sympathetic irritation developed after implantation, but was "cured by removal of the implanted globe."

This was the only case of sympathetic trouble in de Schweinitz's[1] report of 72 implantation operations by 15 American operators, with the escape of the ball in 15 cases. No detailed history of this case is given, and presumably the operator attached little importance to it, as in a subsequent paper[2] on the value of the Frost-Lang operation he makes no mention of this case, and says: "It will be found that in these cases, where sympathetic irritation follows either scleral or capsule implantation, the wearing of a shell is frequently a sufficient cause for the reaction."

[1] "Comparative Value of Enucleation and the Operations which have been Substituted for it." de Schweinitz, xiii. Internat. Oph. Cong., 1900.

[2] *Trans. Amer. Acad. Ophth. and Oto-Laryng.*, 1907.

Since the summary of de Schweinitz, I have found only two instances of sympathetic trouble occurring in cases in which the implantation operation was performed, and this agrees with Gifford's analysis of the cases of sympathetic disease following the various substitute operations for enucleation.

CASE IV.—Reported by Sattler.[1] Injury from bird-shot, followed by a low-grade, painful iridocyclitis with recurrent hemorrhages. Enucleation and glass-ball implantation fifteen days after injury. Left hospital twenty-five days after operation. Shortly afterward complained of transitory dimness of vision, at first at long intervals, but becoming more and more frequent, finally resulting in complete helplessness sixty-two days after the original injury. The ophthalmoscope showed hazy aqueous and vitreous, deposits on Descemet's membrane, and typical papillo-retinitis. The glass ball was removed and 2½*cm* of the optic nerve resected, the cut nerve being found firmly surrounded and embedded in dense cicatricial tissue. Forty days after resection of the nerve the papillo-retinitis of the eye subsided and vision was restored to 0.6.

In Gifford's analyses[2] of the cases of sympathetic disease following the various substitute operations for enucleation he refers to the case of Sattler, but excludes it from a group of 7 cases to which he states that it does not seem reasonable to apply the explanation that the sympathetic ophthalmia might have occurred just the same even if a simple enucleation had been performed. It is difficult in this case to indicate the exact factor causing the sympathetic trouble. Whatever it was, the process was extremely slow, the attacks of dimness of vision not being accompanied by external inflammatory phenomena. It may be that the dense cicatricial tissue in which the cut optic nerve-trunk was surrounded and embedded may have been a factor causing the disease, or it is not impossible that the sympathetic disease may have started before the eyeball was removed, and slowly developed during the twenty-five days the patient was in the hospital after the enucleation, and in the three weeks fol-

[1] *Trans. Amer. Ophth. Soc.*, vol. x., 1904.
[2] *Trans. Amer. Acad. of Ophth. and Oto-Laryng.*, 1908.

lowing his discharge to the time his sight failed. It is hardly possible that the glass ball could have exerted pressure on the nerve.

CASE V.—Reported by Davis.[1] Implantation of a paraffin ball in Tenon's capsule one year before examination by Davis. Comfort for ten months until the artificial eye began to turn in and the other eye became congested and painful and continued so for six weeks. Operation showed the paraffin to have escaped from Tenon's capsule and lodged in a solid mass at the posterior end of orbit. Upon its removal the symptoms disappeared.

The sympathetic irritation in this case was due, in the opinion of Davis, to the pressure on the optic nerve of the mass of paraffin which had escaped through an opening in the capsule made by faulty technic on the part of the original operator. It is possible that the irregular pressure and dragging on the orbital tissues by the displaced artificial shell was also a factor in the cause of the symptoms, the irritation being of a character similar to that which occurs in the sound eye from the wearing of a badly fitting artificial shell.

One case included in the series reported in this paper is of sufficient interest to warrant an extended notice:

Dr. J., while treating a patient in September, 1906, became infected in the left eye with gonorrhœal pus. There was the usual intense conjunctival chemosis, with extensive corneal ulceration, and, after subsidence of the inflammation, dense scarring of the cornea. In December, 1906, staphyloma of the cornea appeared, with secondary glaucoma and perforation of the cornea. The globe was removed at that time, and a 13*mm* gold ball implanted in Tenon's capsule. No reaction after operation; the wound healed by primary union, and in three weeks the patient commenced to wear a reform artificial eye. There was no irritation of the stump, and he had perfect comfort in the use of the right eye until December, 1909, at which time he broke the artificial eye which he had worn for three years. He then secured a new prosthesis, and later two additional shells were made to

[1] Section on Ophthalmology, Amer. Med. Assoc., 1907.

order, but he found that any prolonged reading or close work was followed by pain in his right eye and considerable palpebral injection. Under rest of the eyes and local applications the irritation disappeared, to return again with the use of the eyes at close range. The artificial eye was as comfortable as the one previously worn; there was no discharge from the socket nor congestion due to the wearing of any of the three new eyes, nor was there any tenderness upon pressure upon the implanted ball. The right eye was perfectly healthy, and vision equalled $\frac{6}{6}$. The irritation presented the characteristics of a catarrhal inflammation, with congestion of the lids and of the superficial conjunctival vessels, particularly over the external rectus and slightly over the internus, and extending to the limbus. The man's general health is good, although he is under a heavy strain with his private work and in operative work in a large general hospital connected with a big steel works.

I did not believe that the irritation of the right eye was in any way due to the presence of the implanted ball, nor did it seem possible that the comfortably fitting reform eye could cause the trouble. This was also the opinion of Dr. de Schweinitz and Dr. Hansell, who examined him. It was recognized that the depression in the man's general nervous system through inability to do his close work, and the fear of serious disease to his remaining eye, would compel removal of the gold ball unless the cause of the trouble could be located and the symptoms relieved. Examination of the nose and throat and accessory sinuses was negative. Internal remedies were given to correct a possible rheumatic condition, a slight change was made in the axis of his astigmatic glass, and a weak convex lens was given in addition for near work to lessen accommodative strain, but little if any relief followed these measures. Two reform eyes were made of slightly less width and breadth than those previously worn, so as to relieve any possible pressure at the periphery of the socket, and, while he has been in much better health, and has gained in weight since his return home, there is congestion of the eye, but no pain, the morning following late hours the night before, particularly after smoking one or two cigars

or reading fine print. The eye is not affected at all by operative work or general office work during the day. This is the latest report I have had, dated one week ago.

Measurements were made in many of the cases of the amount of movement of the artificial shell, and the average in 12 cases showed rotation upward of 23°, downward of 40°, inward of 21°, and outward of 19°. Little value can be placed upon measurements of rotation, as they depend so largely upon the shape and fit of the artificial eye. In several instances there was marked movement of the conjunctival bed and yet only moderate rotation of the artificial shell. In one case there was a difference of from 3° to 4° in the extreme excursion of two shells that were both comfortable and yet were slightly different in shape.

TUMOR OF THE ADRENAL GLAND WITH METASTASIS IN THE ORBIT.[1]

By Dr. ALEX. QUACKENBOSS, Boston, Mass.

Pathologic Examination by Dr. F. H. VERHOEFF, Boston.

(*With one figure on Text-Plate XVI.*)

DURING the past three years two cases of tumor of the adrenal gland with metastasis in and about the orbit have come under my care at the Massachusetts Charitable Eye and Ear Infirmary. The first case, a male infant, aged sixteen months, was seen in September, 1906, and was reported in the *American Journal of the Medical Sciences*, June, 1908, by Drs. Tileston and Wolbach.

The second case, and the one which I wish to report, is that of a little boy, three years of age, of French-Canadian parentage. He was brought to the Infirmary in November, 1909, with the story that, about one month before, the lids of the left eye became discolored and the eyeball prominent. There was no history of injury, and, so far as we were able to make out from the parents, the child had always been well.

The upper and lower lids of the left eye are discolored and have the appearance of an ordinary hemorrhage into the lids; there is moderate exophthalmos, the eye being pushed forward and upward. Motion is somewhat limited. The pupil reacts, the vision is apparently good, and the fundus is normal. In the lower-outer part of the orbit a distinct tumor is felt, which seems to be adherent to the

[1] Read at the meeting of the American Ophthalmological Society, Washington, May, 1910.

To Illustrate Dr. Alex. Quackenboss's Article on "Tumor of the Adrenal Gland with Metastasis in the Orbit."

Metastatic neuroma malignum, showing rosettes which represent atypical embryonic ganglia. Photo. x 126.

bone, and just below, a round bony growth is made out, as if the antrum was involved. The right eye is normal.

Dr. Tileston kindly looked the case over and made the following report: In the right side of the abdomen is a large mass reaching from the margin of the ribs to *2cm* below the navel, and from the extreme flank to the median line. The mass in places is quite hard and nodular. Toward the median line the mass is indurated. The spleen is not enlarged. The heart and lungs are negative. The testes are normal. The preauricular glands are not involved; the glands of the neck are enlarged to about the size of a large pea; both anterior and posterior triangles are involved. The glands are more numerous on the left side. The glands in the axilla and groin are slightly enlarged. The reflexes are normal.

To establish the diagnosis, a piece of tissue was removed from the orbit, and at the same time Dr. F. L. Jack removed portions of the growth from the antrum.

This case belongs clinically to that class of adrenal tumors in children in which there is metastasis to the skull—the so-called Hutchison type. The symptoms, which are usually found only in infants and young children, are very characteristic: Ecchymosis of the eyelids, accompanied by exophthalmos, suddenly appears; it may be followed by growths about the orbit. It is usually confined to one side. The glands throughout the body are apt to be enlarged. Examination of the abdomen may reveal a tumor in the region of the kidney, or a mass may be felt in other parts of the abdomen. The blood shows secondary anæmia. The urine is usually free from blood and albumin. Most of the reported cases have been under four years of age. The course of the disease is rapid, death resulting from cachexia and anæmia.

In briefly reporting this case we wish to call attention to the following facts:

(1) Tumors of the orbit in young children, especially if accompanied with ecchymosis of the lid, should arouse suspicion of metastasis from an adrenal growth.

(2) The microscopic examination of specimens shows a characteristic rosette formation of cells.

(3) This form of tumor arises from embryonic nerve-cells of the adrenal gland.

Pathologic Examination.

The specimens submitted for examination consist of curettings from the antrum of Highmore and a small piece of tissue removed from the orbit. Fixation in Zenker's fluid.

On microscopic examination the curettings are found to consist chiefly of bone. The medulla shows an overgrowth of fibrous connective tissue and is invaded by tumor-cells. The bone in places is also attacked by osteoclasts. The tissue has been so severely traumatized that, for the most part, the exact character of the tumor-cells is obscured. They usually occur in bundles of elongated cells which stain with great intensity, the individual cells giving the appearance of nucleated fibrils. This appearance, no doubt, is partly or wholly artefact. In a few places, however, where the tissue has, fortunately, escaped injury, cell structures occur which at once reveal the nature of the tumor. These are groups of cells arranged in the form of so-called rosettes. The typical ones are almost perfectly round, with nuclei at the periphery arranged circularly about a central area of eosinophile material. This material on superficial examination appears to be finely granular, but more careful examination shows that it consists of fine fibrils cut across. In favorable sections the origin of the fibrils from the peripheral cells which abruptly taper into them can be made out. The nuclei are round, have chromatin nets not unlike those of lymphocytes, well-marked nuclear membranes, and show definite nucleoli. They are similar in appearance to the nuclei of the so-called glioma retinæ. These rosettes, however, are entirely different from the rosettes of retinal tumors, because in the latter the cells show a limiting membrane bordering on a lumen.

Mitotic figures are not encountered, owing, no doubt, to the limited amount of material available for examination.

The small piece of orbital tissue removed is also badly traumatized, but shows invasion with cells similar to those in the malar bone. No rosettes are found here.

DIAGNOSIS.—Metastatic neuroma malignum of malar bone and orbit, probably primary in adrenal gland.

REMARKS.

The histologic diagnosis in this case rests chiefly on the presence of the rosettes, which, fortunately enough, are present in some of the specimens. These are identical n every way with those in the tumor of the adrenal gland, metastatic in the orbit, described by Tileston and Wolbach, sections of which I have at hand. This tumor was regarded by Wolbach as a sarcoma. J. H. Wright, however, in a communication which will soon be published, pointed out that this tumor undoubtedly arose from embryonic nerve-cells of the adrenal gland, the rosettes representing atypical embryonic ganglia, and he has reported other cases of his own which he formerly regarded as sarcomata. He finds that two other cases reported in the literature as gliomata are really of this same nature. As a designation for these tumors he has adopted the term *neurocytoma*. This, I think, is too restrictive.

While differing in details, these tumors are similar in general nature to the so-called gliomata of the retina, for which I have suggested the name *neuroma retinæ malignum*.[1] My reason for selecting the general term *neuroma malignum* was that it would be applicable to analogous tumors which might occur elsewhere in the nervous system. The selection of this term has thus been justified. The occurrence of these tumors of the adrenal gland lends support to the view, which I have also advanced, that the cells usually predominating in the retinal tumors correspond to the bipolar cells of the inner nuclear layer, which cells are believed by many to be analogues of the cells of spinal ganglia.

[1] *American Ophthalmological Transactions*, 1904.

STUDIES CONCERNING HEMERALOPIA.

By Prof. CARL HESS, Wuerzburg.

Abridged Translation from the German Edition, Nov., 1907, by Dr. Matthias Lanckton Foster.

I.

SINCE it was demonstrated nearly thirty years ago that hemeralopia was due to a disturbance of adaptation, the idea has gained prevalence in ophthalmological and physiological circles that night-blindness is to be referred mainly or exclusively to the rods of the retina. Among the diseases of the eye on which Parinaud based his hypothesis of a double retina, the principal place is given to hemeralopia, which, according to him, consists of an isolated disease of the rods, or of an absence of visual purple in them. His followers likewise maintain that in uncomplicated hemeralopia the lesion is confined to the rods of the retina. During the past year I have studied several cases of night-blindness and sought to recognize accurately all known forms of hemeralopia. I studied cases of congenital and acquired idiopathic hemeralopia, the forms met with in retinitis pigmentosa and in the course of hepatic disease, as well as that known as "Spring hemeralopia" from its peculiar clinical characteristic.

In order to estimate rightly the symptoms in such patients it seemed desirable to make clear how an eye must see, in which the capacity for the formation of purple, in the sense used by Parinaud, is quite absent and the capacity for dark adaptation wanting, while in all other respects it is normal, particularly as regards the cones, which, according to Parinaud's theory, have no capacity for adaptation. It is a matter of indifference whether such a total absence of adap-

tation is met with in reality or not. First, according to such a theory the foveal vision of the night-blind can be distinguished from that of the normal by a sojourn neither in the light nor in the dark, because the rod-free area is incapable of adaptive changes and is not affected by the disease. Second, in such an eye after a prolonged stay in the dark a lessened sensitiveness of the fovea could not be demonstrated, because in the normal eye this is caused through the great adaptive increase of sensibility of the portion of the retina containing rods. Third, colored lights with decreasing irradiation could not become colorless for such eyes, as they do for the normal eye; on the contrary, they must appear colored to the night-blind eye as long as they can be seen. The freest colors become veiled more and more with white and finally become colorless for the rod-containing retina of the normal eye with the decrease of the light strength in consequence of dark adaptation. According to Parinaud, the explanation of this would be that with correspondingly reduced irradiation the colored lights can act no longer on the cones, but act on the rods made sensitive to light by accumulation of visual purple. If this action on the rods is absent in the night-blind the tendency of colors to become whitish in dark adaptation must be absent also. Fourth, the hemeralopic eye must respond like the normal to free red lights; for the adaptive increase of sensibility through formation of the visual purple is ***nil*** for red light, according to Parinaud, as it acts wholly on the cones, which are held to be normal. It is also claimed by Parinaud's followers that in uncomplicated hemeralopia there is no loss of sensitiveness for red light, therefore with decreasing irradiation the night-blind should see it as long as the normal eye. Fifth, the behavior of the hemeralopic eye to high light strengths may be of interest. It is held that only the cones have high degrees of sensitiveness, while the rods, even with strong stimulation, do not give the impression of greater brightness, but only of a moderately bright gray. In an affection confined to the rods the hemeralopic eye should behave to high-light strengths the same as a normal eye. Schirmer has advanced the theory that slight intensities of light act upon the hemeralopic eye in the same way as greater intensities upon the normal eye, thus a hemeralopic eye may

be abnormally sensitive to light; the photophobia which he claims is so frequently met with among hemeralopes supporting this view.

We shall see that of all these presumptions not a single one corresponds to the facts found in our cases of hemeralopia: in no case have I found the rod-free area even approximately normal, but without exception diseased in the same sense as the extrafoveal retina. Lights to be perceived with the fovea of the night-blind must have greater light strengths than are necessary for perception with a normal fovea equally adapted to the dark. A diminished foveal sensitiveness in the dark and in reduced light was absolutely demonstrated in all the patients who had sufficient powers of observation. Colored surfaces became colorless to the hemeralopes in reduced light, even when the light strength was such that the colors were still distinct to the normal eye equally adapted to the dark. The freest possible red lights became with decreasing light strength invisible to the hemeralopes, even in the central area of the retina and after remaining many hours in the dark, sooner, and in some cases very much sooner, than to the normal. Finally, I found in almost all the night-blind distinctly diminished sensitiveness to very high-light strengths. The usual theory that hemeralopia can be ascribed to an absence of the visual purple as a mediator of the dark adaptation in the sense used by Parinaud is thus contradicted by all the facts here observed.

The hypothesis that a dark adaptation caused by the visual purple is present in hemeralopes, but of less extent than in the normal, necessitates that the fovea be normal, but the fact is that in all the cases investigated by me there was a characteristic disturbance of the foveal vision; likewise it cannot explain the reduction of the sensitiveness of hemeralopes for high-light strengths that I so often found. Under the same conditions the hemeralope should perceive the color of a colored light, when the room is darkened, as long as the normal, but we shall see that this is not the case: the colored lights appeared colorless to the night-blind when, under otherwise like conditions, the color was still distinct to the normal eye.

Finally, as regards the diminished foveal sensitiveness for faint lights after remaining in the dark for a long time. Pari-

naud and his followers maintained that the cones in hemeralopia were normal, and that the normally rod-free area of the retina was incapable of adaptive changes. According to this untenable theory, a diminished foveal sensibility can be found in night-blind eyes after a long stay in the dark, when the rod-containing portion of the retina shows only a relatively small fraction of the normal capacity for adaptation, but the fact is as established by Hering, that the rod-free area is capable of marked adaptive changes; it differs from the rod-containing only by its incomplete adaptation to the dark.

With the theory that in the night-blind eye the cones adapt normally and only the rods faultily for dark, we had to take into account the following possibilities: First, the capacity for dark adaptation is lowered in the rod-containing area, but so little that after a sufficiently long stay in the dark a foveal hyposensibility still exists; the difference between the sensibility within and without the rod-free area is then less than in the normal eye adapted to the dark for the same length of time. Second, the dark adaptation of the rod-containing retina is so far lowered that in a certain stage the rod-containing and the rod-free portions of the retina are approximately equally sensitive when the foveal hyposensibility of the dark-adapted eye equals 0. Third, the adaptation capacity of the rods is so faulty that after a long stay in the dark the rod-containing area, in which the cones are much less dense than in the fovea, may be less sensitive to light than the rod-free. In such a case there might be either no difference between the rod-containing and the rod-free retina, or a foveal hypersensibility, but there could be no foveal hyposensibility of the dark-adapted eye.

In all the cases of night-blindness I examined, the rod-free area of the dark-adapted eye was distinctly less sensitive than normal. Either such anomalies confined to the rods as were assumed by Parinaud were not present, or all ten of the cases I examined belong to another group which differs from that of Parinaud by a more or less marked involvement of the cones in the disease. The latter is hardly probable. It will not be difficult to determine by further study whether there are hemeralopias with normal cones.

II.

In accordance with the views just laid down I have developed a method of investigation of the night-blind which I will describe in connection with the histories of ten cases of hemeralopia that I have studied carefully.

CASE I.—Mr. H., 52 years old. Acute hemeralopia with hepatic disease. The patient had been treated five months before for jaundice. A laparotomy, because cancerous occlusion of the ductus choledochus was suspected, revealed nothing positive; the gall-bladder was joined to the intestine; recovery was uneventful. He has complained for some weeks of bad vision in the evening. Vision of each eye $\frac{6}{6}$ with +1.0 D. Fundus normal.

The night-blindness was first demonstrated by examination in a dark room provided with an Aubert's screen. In this and in the following cases nine dull-white surfaces 1*cm* square, in three rows and from 6 to 8*cm* apart on a black background, were used as objects of vision at a distance of 40 to 50*cm* from the eye; some of the observations were made with, others without, correcting lenses. While the light was sufficiently strong, the patient directed his eye to about the centre of the group of white surfaces and the opening in the screen was then gradually diminished until the squares were invisible. The following figures give in millimetres the side lengths of the square opening in the screen.

The limit of visibility of the white surfaces for one or two minutes after entering the dark room is reached with 7*mm*. H., even with the maximal opening, 45*mm*., after remaining from 10 to 15 minutes in the dark, sees nothing of the white surfaces with his right eye, and just as little with his left, which has been covered with a bandage impervious to light for 12 hours. During the frequent repetitions of this experiment, he stated off and on, uncertainly, that he could see something with 30*mm*, but then with 35–45*mm* he also saw nothing of the surfaces, which appeared fairly bright with this opening to my eye, which had been adapted to the dark for only a short time. For myself, after remaining in the dark for about 10 minutes the limit of visibility of the disks was at 5*mm*, though during this time I did not keep my eyes closed and repeatedly looked at the bright diaphragm.

This and similar experiments go to show that the theory is at least not of general application according to which night-blindness consists in the great majority of cases entirely

of a delayed dark adaptation, and that with a sufficienty long stay in the dark—12 hours—the same sensitiveness to light can be attained as in the normal eye.

Ten days later the patient stated that he could see better in the evening. In an examination without previous bandaging of one eye, and in which we entered the dark room together, the limit after one or two minutes was *7mm* for me, 30 to 40*mm* for him. A bandage impervious to light was then placed over his right eye. After about 20 minutes the limit for me was at 5*mm*, for his left eye at 35 to 40*mm*. After his right eye had been bandaged 45 minutes, the limit for it was at 13*mm*., for me at 4–5*mm*.

Five days later he stated that he could see much better in the evening than formerly and better with his right eye than with his left. The right eye was again bandaged for 12 hours. Then we entered the dark room together. The limit for his left eye was at 30 to 35*mm*, for his right the same as for me, 5–6*mm*. I now tried together with the patient to look at the bright sky and found it very difficult. He stated that it was uncomfortable for his right eye, which had been long adapted to the dark, yet he could keep it open without apparent trouble; with the other eye, which had been adapted to the dark for perhaps a quarter of an hour, the bright sky appeared less bright and he had no sensation of dazzling. Some days previously he had been able to look at the sun without trouble after remaining a long time in the dark. After looking at the bright sky for some minutes the difference between his right and his left eyes was not so great, but was still present; the sky appeared brighter and more yellowish with his right eye. We then returned to the dark room. The limit of visibility of his right eye again agreed fairly well with mine; that for the left was at 35 to 40*mm*.

Among other experiments the following was made concerning the color vision of the patient in reduced illumination with his eyes adapted to light and dark. Large colored surfaces were moved slowly back and forth in the dark room before the eye of the patient standing with his back to the light, at first with so weak an illumination that he saw nothing. The opening in the screen was then gradually opened until he could perceive a movement. With a light strength in which, under otherwise the same conditions, I could distinctly see the colors, he pronounced the yellow, green, and blue surfaces to be "somewhat gray, colorless"; the light strength had to be increased considerably to enable him to see the colors; at first they appeared

to him as gray green, gray blue, etc.; with sufficient light he saw them clearly. A red surface he called reddish gray, gray, or colorless when the light was so reduced that it appeared nearly colorless to me; with more light he called it red; with repeated reduction of the illumination he said that certain parts of the surface still appeared reddish while other parts were purely gray, "without red." With a spectrum, using a considerable amount of light, the limit at the red end appeared to be about the same for us both; he could see the red, yellow, and green, but not the blue and violet. He indicated the limit toward the short-waved end of the spectrum in the region of blue. When the light strength was slightly reduced he saw the region of green as distinctly bright, but no longer green, although to me it still appeared green; at the same time red still appeared red. When the light strength was still more reduced, I could not determine that red appeared colorless to him; it rather disappeared, but he no longer saw the red of the spectrum, while it was still clear to me.

It was proven experimentally that a lessened sensibility to blue as the result of jaundice or a yellow lens was not present.

These experiments show that the rod-free area of the retina of his dark-adapted eye was much less sensitive than normal to faintly lighted stimuli. To forestall the objection that perhaps the line of fixation had not been maintained with sufficient certainty, I arranged a number of round white disks on a black surface in vertical and horizontal rows and directed the patient to look at the centre. With sufficient light strength the disks seen foveally and extrafoveally appeared to him equally bright, but as the light was reduced the disks seen with direct vision seemed less bright, and when it was reduced still more they became invisible, though the rest were still clearly seen, while to me they appeared very distinct and not less bright than those seen indirectly. With the illumination still further reduced all the disks disappeared to him, although they were all still distinctly visible to me.

The difference for red between his eye and mine was less, but even then the disks seen foveally and extrafoveally disappeared completely for him with reduced illumination, when they still appeared distinctly red to me. This was repeatedly the case even when his eye had been adapted to the dark for 12 hours. With blue the difference was much greater; he could see nothing any longer at a time when all the disks looked very blue and comparatively bright to me.

There was no contraction of the visual field when the

investigation was made with sufficient light to permit him to see the object in the perimeter foveally; repeatedly he was able to see indirectly the object far toward the periphery when he could not perceive it in the neighborhood of the point of fixation.

Purkinje's phenomenon was studied in the following manner: A blue square of *2cm* side length was placed on an even red surface; with a strong light the patient saw the surface to be red, the square blue; with the illumination reduced so that I could still see the red and the blue distinctly the background still looked red to him, while the square appeared darker than the background when he looked at it directly; with indirect vision it appeared distinctly brighter to him.

When a strip of blue paper 7 or 8*mm* wide was placed over the red surface, he repeatedly stated that when the light was reduced it appeared to be a colorless gray strip on a dark background, and the place in the strip directly fixed on seemed darker than the rest.

Ten days after the last examination he stated that for the past two days he had been seeing worse again in the evening. Light was again excluded from his left eye for 12 hours. Examination of his right eye with Aubert's screen gave the limit of visibility of the white square at 20*mm*, for me at 4*mm*. With his left eye long adapted to the dark he could see even with 30*mm* only 2 or 4 of the squares in the outer-lower portion of the visual field. Even with 43*mm* he could not see all of the white squares, though he could with the other eye. Red disks on a dark background became invisible for both his eyes, as the illumination was decreased, much sooner than for mine.

About four months later the jaundice and the hemeralopia were considerably greater, the vision in the light unchanged. With methods that I have described elsewhere I demonstrated that now the light strength necessary to make just visible a white surface looked at through a dark ruby-red glass was 80 to 100 times greater for him than for me. A red and a blue surface were invisible to him when visible to me; as the light strength was increased, the blue surface appeared gray to him, and he said it appeared darker than the other, which seemed slightly reddish.

Case II.—Herr F., 64 years old. Idiopathic (?) hemeralopia. Vision of each eye $\frac{6}{6}$. Fundus normal. The patient, who appeared somewhat jaundiced, said that he had been seeing badly in the evening for about four weeks. He had a slight enlargement of the left lobe of the liver and nephritis.

In the examination with Aubert's screen the white

squares disappeared at 27*mm*, for me at 5 or 6*mm*. After ten minutes' adaptation to the dark they disappeared at 20*mm*. A dressing impervious to light was placed over his right eye for 17 hours; on the following morning the limit of visibility of the white squares was at 20*mm* for his left eye, at from 15 to 20*mm* for his right, at 4 to 5*mm* for me. A more accurate determination of the limit was impossible, because with the reduction of the illumination all the squares did not disappear simultaneously, but those in the outer-upper part of the field regularly remained visible longer than the others. Repeatedly he said he could see only one square, always the one farthest to the right and above, even after the squares had been changed about so as to exclude the possibility that one was of greater light strength than the others. Such a regional inequality of dark adaptation I noticed repeatedly in the patient first mentioned.

When the patient used his right and his left eye alternately to look at the squares with the opening in the screen at 20 or 25*mm* he said that they seemed scarcely any brighter with the one from which light had been excluded for 17 hours than with the other. Hence in this case also exclusion of light even for 17 hours produced no noticeable increase in the sensitiveness of the eye.

In all other points the results of the examination of this patient coincided with those obtained in the first case.

CASES III. and IV.—W. Joh., 20 years old. High degree of congenital (idiopathic?) hemeralopia with xerosis. The patient had always seen very badly in the evening, so much so that he was helpless in the dusk. The conjunctiva of each eye in the region of the palpebral fissure appeared dry, to a greater extent outward than inward, and the temporal portions of these dry parts were covered with a fine white froth. Vision was $\frac{6}{6}$. Fundus normal.

Examination with Aubert's screen showed that all the white squares were invisible to his right eye two or three minutes after entrance into the dark when the aperture had a side length of 45*mm*, while for me, after an equally long stay in the dark, the limit was at 3*mm*. In some of the other examinations he saw some of the squares uncertainly after remaining in the dark for a short time. The right and then the left eye were alternately bound up for twelve hours several times; the limit of the visibility of the disks remained the same as before at 45*mm*, showing that in this case also there was no perceptible adaptative increase of sensibility. Once he said that the squares appeared to

him perhaps a trace brighter with the eye that had been bound up than with the other.

When he looked at the white disks on a dark background he said that the disks fixed upon at any time appeared less bright than the others which were seen excentrically.

His limit toward the red end of the spectrum was about the same as mine, but toward the short-waved end his limit was in the blue green. The spectral green he called, even with considerable light strength, colorless gray. Even with the eye adapted to the dark for 14 hours he saw in the spectrum with moderate light strength only red and "gray." With the light strength still more reduced, the red disappeared for this eye much sooner than for mine, adapted to the dark for only a short time.

Colored surfaces appeared colorless to him when the light strength was decidedly greater than in the preceding case. Even red he called dark gray when it still appeared red to me. In the examination with the arrangement of bright disks upon a dark background, all the disks became invisible to him even in the rod-free area with reduced illumination, at a time when they still appeared so bright to me that the foveal hyposensitiveness of the normal eye was not brought out. The difference between us was again most marked with the blue disks, but red also became invisible to him much sooner than for me. In comparison between an eye relatively adapted to light and one adapted to dark for half an hour, he said once that with the latter he could still see the disks in an illumination in which he could not see them with the other.

Hence in this patient with congenital night-blindness the foveal sensitiveness of the eye was much less than normal in reduced illumination; there was a distinct hyposensitiveness of the rod-free as compared with the rod-containing portions of his retina, and even free red lights became invisible to him with reduced illumination much sooner than to me. In looking at the sun or the bright sky he was less dazzled than I.

Exactly the same results were obtained from the examination of another man with congenital hemeralopia, aged 22 years.

CASE V.—Mrs. G., 54 years old. Has had bad vision in the evening for about ten years. Hemeralopia with retinitis pigmentosa. Vision $\frac{6}{12}$ to $\frac{6}{6}$ with +1. Papilla pale, choroidal vessels very distinct, marked retinitis pigmentosa. Visual field contracted temporally to 20–25,° nasally, upward and downward to 15–20°.

With Aubert's screen the white squares disappeared for her at 16*mm*, while under the same conditions the limit of visibility for me was at 4*mm*. In the spectrum she saw with a good light strength red, yellow, green, and blue, the abridgment at the short-waved end less than in the cases already described. As the light strength was diminished, she saw green only as "bright grayish," while red was still distinct; when the light was yet more diminished, red disappeared, though it was still visible to me. In the same way the red disks on a dark background became invisible to her, as the light was reduced, much sooner than to me. A red disk still looked red when a blue one appeared gray. She could look at the sun with scarcely any sensation of dazzling; foveal hyposensitiveness was distinctly demonstrable.

CASE VI.—J. R., 59 years old. Spring hemeralopia. This patient came on May 19th with the statement that for the last five weeks he had seen badly in the evening; this trouble, which he had not noticed in early life, had gradually come on. In other respects he was apparently healthy. Vision in each eye was $\frac{6}{6}$ with +1. On a sunny day the limit of visibility of the white squares with Aubert's screen was at 15–20*mm*, for me at 1–2*mm*. Blue, green, and yellow surfaces appeared to him a colorless gray with reduced illumination when the colors still seemed distinct to me. Red disks disappeared for him, as the light was reduced, much sooner than for me. He was little dazzled by looking at the sun. The test for the foveal hyposensitiveness was unsatisfactory because of his lack of attention. In the following cases I could not prosecute the examination in all directions as I desired.

CASE VII.—H., 20 years old. Idiopathic hemeralopia. Has seen very badly in the evening since childhood. V. o. d. $\frac{6}{9}$ with − 6. Broad conus. Field normal.

All the white disks on a dark background became invisible to him, as the light was reduced, much sooner than to me. The disk fixed on foveally became quite invisible when the light was such that my foveal hyposensitiveness was not perceptible. With indirect vision a small group of points appeared somewhat hazy, but brighter than that seen with direct vision. After remaining a long time in the dark he could look directly at a very strong light which dazzled me. The spectrum appeared much abbreviated toward the short-waved end. Looking at a red and a blue surface which seemed to me to be about equally bright by daylight, the blue seemed brighter than the red as the light was reduced. When the light was still more reduced, both surfaces disappeared much sooner than they did for

me. Of two blue disks seen on a dark background, the one seen indirectly seemed much the brighter. As the light was gradually reduced, the one seen directly disappeared, while the one seen indirectly still appeared blue. When he looked at the bright sky through a free blue glass he immediately saw his macula as a dark spot. A blue band on a red ground seemed much darker at the place fixed upon than elsewhere. When he looked at the sun he had a slight feeling of dazzling and distinct after-images.

CASE VIII. — Mr. M., 36 years old. Hemeralopia with retinitis pigmentosa. Vision of each eye $\frac{6}{12}$ with +1. Marked pigment atrophy; choroidal vessels very distinct.

White disks on a dark ground became invisible when the light was still so strong that my foveal hyposensitiveness could not be perceived. He stated without being asked that formerly when he looked at the sun he had seen distinct after-images when he closed his eyes, but now he could not perceive them and he could look at the sun without the least trouble. This statement was experimentally confirmed. When light had been excluded from one eye for a quarter of an hour and then a faintly illuminated white disk on a dark background was looked at through a prism so as to produce double images the two images appeared equally bright, just as they had previously with two open eyes in the light, but after light had been excluded from one eye for 14 hours the disk seen with that eye seemed much the brighter. In clear daylight he pronounced a red surface distinctly brighter than a blue. When the light was reduced both became colorless, the red almost black, much darker than the blue.

CASE IX.—Chr. B., 21 years old. Idiopathic hemeralopia. High degree of night-blindness since childhood. After remaining long in the dark he could look at the sun or a very strong arc light without trace of dazzling. As illumination was reduced, red and blue surfaces became invisible when the blue still appeared to me distinct and blue. All the white squares disappeared for him much sooner than for me.

CASE X.—Mr. S., 54 years old. Spring hemeralopia. Vision $\frac{6}{6}$. Fundus normal. I saw this patient first in 1902 when he said that since 1895 hemeralopia had suddenly come on each spring after one or two days of burning of his eyes and continued for 5 or 6 weeks. During the past year it had lasted 2½ months. From another patient I also obtained the history that for several years he had had hemeralopia each spring for some time. Accurate

examination was impossible; I could only demonstrate the great reduction of sensitiveness to light as the illumination was diminished, the abridgment of the spectrum at the short-waved end, and the fact that he had no trace of photophobia.

III.

More than 40 years ago Max Schultze advanced the view that the rods were concerned in the sense of light and space, the cones in the color sense. Since Parinaud has developed his theory that the rods alone possess the capacity for adaptation, in which the cones are completely wanting, it has been assumed that under fixed physiological relations and with certain pathological conditions of the eye one component of Parinaud's double retina functionates preponderatingly or exclusively while the other is more or less completely excluded. This has long been supported by assertions concerning the vision of the totally color-blind, the vision of day birds, and the vision of the night-blind. That the vision of the totally color-blind is not to be considered as an isolated activity of the rods, as a rod vision, is proven by the studies of Hering and myself. That hens and doves are not night-blind and that their retinæ are capable of adaptive changes in spite of the almost complete absence of visual purple I have already shown, and finally the studies here given of all known forms of hemeralopia show that night-blindness is not due to a lesion confined to the rods. In all I have found in the central part of the retina, which contains cones alone, disturbances of the same kind as have hitherto been assumed to be present only in the parts containing rods. An isolated disease of the latter was not present in a single case.

This study of hemeralopes shows impressively the difference between the vision of a normal fovea and that of one nearly or quite incapable of adaptation. Hering has shown that the visual purple can be demonstrated even when attenuated, and the explanation of the vision of the night-blind might be that purple was present in the normally rod-free area, but in too small quantities to be perceived, and that even these small quantities of purple are more or less absent

in the retina of the night-blind. But even so the facts are not sufficiently explained.

As my studies of the vision of day birds has shown the presence of adaptive processes independent of the visual purple, we must consider the possibility that night-blindness is due to the failure, or the congenital absence, of the capacity for such changes in the visual epithelium, particularly in the cones, with which the accommodation of the eye to different light strengths is united.

Whether a faulty formation of visual purple plays a part in night-blindness or not, we do not know. Since I have shown the way to fix with fair accuracy the dark adaptation of the normal eye in monkeys, hens, and doves, it is not impossible to approach this question experimentally, to produce disease of the liver, for instance, and then test the behavior of the dark adaptation and the visual purple in such animals.

In another article I propose to give the methods by which the differences between the color vision of the normal and the night-blind in reduced illumination may be made accessible to treatment, and shall describe a kinesimetric procedure to demonstrate hemeralopia objectively, independent of the statements of the patients, and to measure it within certain limits.

Supplement.

The patient Hoe died about four weeks after my last examination. His vision in daylight was still good shortly before death. The autopsy revealed partial occlusion of the cystic duct by carcinoma, great dilatation of the intrahepatic biliary ducts, and callous thickening of the walls of the latter. There was yellow atrophy of the liver; the meninges, sclera, optic nerve, and other organs were stained yellow. It was impossible to protect his eyes sufficiently from light just before and after death, as I had wished, in order to study the behavior of the visual purple. I found the retinæ which had been prepared with the greatest practicable protection against light perfectly colorless, and could perceive no difference in the stain between one which had been exposed to the light for some time and the other which had been kept in the dark.

CLINICAL STUDIES OF THE VESSELS OF THE CORNEA.

By Dr. A. BRUECKNER, Wuerzburg.

Abridged Translation from the *Archiv fuer Augenheilkunde*, Nov., 1908, by Dr. Matthias Lanckton Foster.

(With figures 1–28 on Text-Plates XVII.–XXIII.)

FOR the study of the corneal vessels I have used almost exclusively Zeiss's binocular loupe, usually with objective a_2 and ocular 2, which together give an enlargement of 24.

I.—General Portion.

1. *Anatomical relations of the corneal vessels.*

We have to distinguish between the superficial and deep loops at the margin of the cornea. The one presents the region of anastomosis between the conjunctival and episcleral capillaries, the hyperæmia of which causes the well-known circumcorneal injection; the others are much more sparse and are invisible during life. The pathological corneal vessels springing partly from these deep marginal loops will be studied first.

a. Course and arrangement of the corneal vessels. The vessels that arise from the superficial loops branch like trees, while those from the deep loops usually run a straight course and show the "birch broom" form, a course due to the arrangement of the corneal lamellæ. Yet this form is not without exception. In a case of interstitial keratitis due to hereditary syphilis, I found at the apex of the palisade-like advancing deep vessels a branching like the top of a tree, which corresponded perfectly with the dendritic branching

TO ILLUSTRATE DR. A. BRUECKNER'S ARTICLE ON "CLINICAL STUDIES OF THE VESSELS OF THE CORNEA."

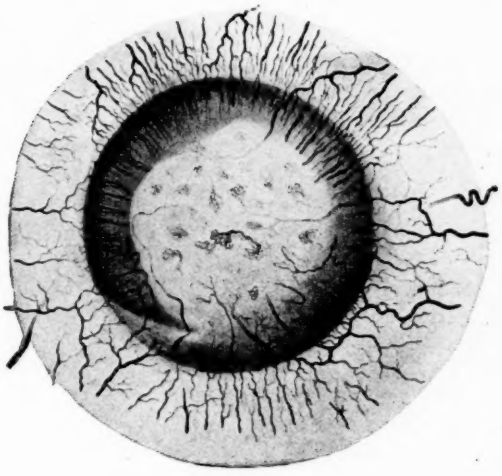


Fig. 1

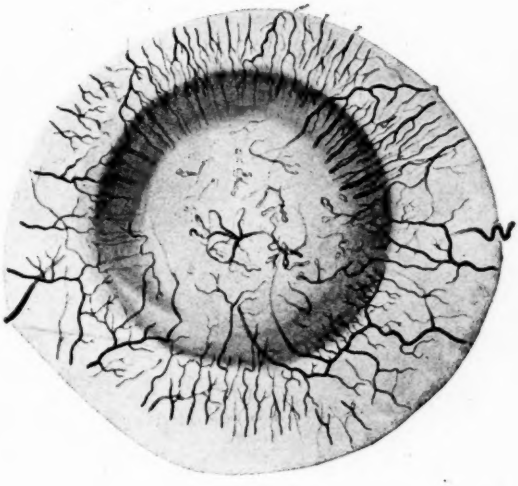


Fig. 2

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of the superficial vessels. After a few weeks this appearance was lost again because the individual loops of vessels extended longitudinally in their further growth and resembled the typical lamellar vessels. While the afferent and efferent stems of the branching superficial vessels, as a rule, follow separate courses, those of the lamellar are accustomed to run parallel. Toward the centre of the cornea they are joined by a short arch so as to form a loop. The calibre of the venous branch is always much greater than that of the arterial, which is frequently invisible when lower powers are used. Hence in several of the illustrations only the venous branch is shown. The immediately neighboring parallel course of the afferent and efferent branches is not always given because, even with great enlargement, only a single vascular twig can be seen. This separation can be so great that the vessels belonging together may be at very different depths, the supposed condition in the case pictured in Fig. 6. This was the case of a seamstress 32 years old who had an obstinate eczematous keratitis. The afferent vessels for the relatively large venous branch *v*, which runs between the anterior and middle thirds of the thickness of the cornea evidently lie among the arterial vessels of the vascular band *x*. The arterio-venous pair of vessels shows a particularly regular parallel course when a finger-shaped splintering of the stem has taken place, as sometimes happens in fresh exzematous eruptions on the margin. Compare Figures 9 and 10, in which the difference in the thickness between the arterial and venous branches appears too slight.

b. Origin of the corneal vessels. Augstein has stated that sometimes a superficial curvilinear vessel passes into the deeper layers of the cornea and then follows the direct course of the lamellar vessels. I can confirm this, but would say that such an occurrence is not very frequent. I have met with it several times in interstitial keratitis and occasionally in eczematous keratitis, as shown in Fig. 11. It can be clearly seen in this picture how vessels from the superficial marginal network above enter the cornea, then penetrate into it and become lamellar vessels. Even then they are tolerably superficial. More frequently such a transition and course is to be seen in corneæ that have undergone an extensive

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cicatricial transformation as the result of ulceration, recurrent eczematous ulcerations, and ulcus serpens as in Fig. 1. In these cases the lamellar structure is no longer present because the structure of the cornea is lost, but therewith disappears the force which usually prevents the penetration of the vessels into other layers of the cornea.

It is not rare that a relatively thick episcleral vessel enters the cornea as a lamellar branch from the beginning, especially in eczematous affections. In these cases the position of the lamellar vessels is relatively superficial (Figs. 5 and 11). If the ulcerative process has been localized at the limbus the connection between the two sections of vessels inside and outside the cornea cannot always be recognized with certainty, in many cases it can only be inferred. But on the other hand cases are to be observed in which the vessel can be clearly distinguished throughout its course.

The most superficial vessels, lying almost subepithelially, as well as the lamellar ones in the anterior layers of the cornea arise usually if not always from the superficial marginal network. But the origin of the lamellar vessels in the middle and deepest layers of the cornea must be sought in the deep marginal loops. The same may serve sometimes for the superficially running lamellar stems when the connection with the anterior marginal network is wanting. With reference to the different depths of the lamellar vessels, either a different depth must be ascribed to the deep marginal loops individually, or for many cases a new formation of vessels must be assumed together with a sprouting of vessels from the deep marginal loops. Probably both occur. In favor of the second suggestion is the circumstance that the deepest vessels develop relatively late, at least as compared with the more superficial. In this case the vessels in the neighborhood of Schlemm's canal may be the place of origin.

c. Anastomoses between vessels of different depths. As an oblique course of vessels in the tissue of the cornea is not so very frequent, it is understandable that anastomoses between vessels in different depths of the cornea are to be observed comparatively rarely. They are more frequent when the cornea is largely cicatricial and its normal structure is destroyed, but otherwise anastomoses of the larger stems are

TO ILLUSTRATE DR. A. BRUECKNER'S ARTICLE ON "CLINICAL STUDIES OF THE VESSELS OF THE CORNEA."

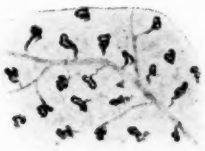

Fig. 3

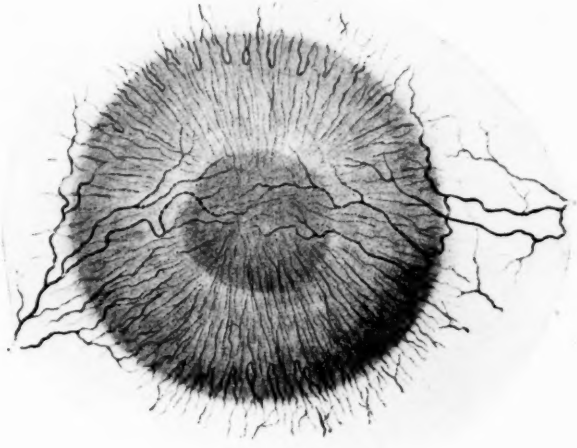

Fig. 4

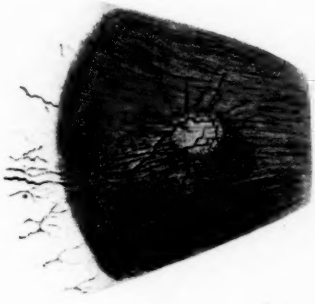

Fig. 5

very rare. I have found only one case of such a union between a subepithelial and a lamellar vessel (Fig. 12) in two and a half years. This was the case of a woman 31 years old who presented a central fresh infiltration together with old superficial spots on the cornea. As the picture shows, a twig from the subepithelial vascular network not far from the centre of the cornea bends backward in order to join a twig of like size of the lamellar network, which lay about on the boundary between the anterior and middle third of the thickness of the cornea.

A similar, but different, anastomosis is shown in Fig. 11. Above are vessels which originate in the superficial marginal network and soon penetrate into the cornea. About in the centre of the latter the anastomosis takes place with other lamellar vessels which enter the cornea in its deeper layers. The anastomosis in this case is between lamellar vessels one of which did not originally have a lamellar course.

Junctions of vessels that arise one from the superficial, the other from the deep marginal, loops are met with in large number only in cases of interstitial keratitis. As this disease is not common about Wuerzburg, I do not know whether the form of vascular connection I found in two cases is regularly present or not. In these the development of lamellar vessels took place in the manner described as usual in this disease, above and below, but from the sides closely set vascular twigs extended from the superficial marginal network upon the cornea beneath the epithelium, where they branched dendritically and formed connections with the intralamellar vessels by means of individual penetrating twigs. Peripherally these subepithelial vessels passed over into the episcleral vascular system, particularly in the veins. Fig. 4 shows this condition without the anastomoses, because this process had begun to pass away and was no longer clearly recognizable when the drawing was made. In the cases mentioned the anastomoses apparently took place between veins, while connections between the arteries have not been demonstrated. The fact that it is more difficult to see the arteries than the veins, because of their smaller diameter, is not sufficient to explain this; it is more probable that there is no anastomosis between arterial stems of different depths.

Because of its rarity I wish to mention here a case pictured in Fig. 27.

A woman 40 years of age had had iridectomy performed two years before on account of secondary glaucoma following iritis. On the temporal leg of the coloboma of the iris was a fairly thick vessel which bent backward about its margin, but peripherally took its way through the entire thickness of the cornea to continue as an episcleral vein. Hence there was in this case an anastomosis between the episcleral vessels and those of the iris by means of the scar left by the operation. Whether this connection was formed immediately after the operation or later is uncertain.

2. *Development of the corneal vessels.*

The development of the superficial, subepithelial vessels has been dealt with so thoroughly by Augstein that I have given it less consideration. While for these vessels a pushing forward of individual sprouts into the cornea appears to be characteristic, it is clinically demonstrable that the lamellar vessels always start as a loop—that is, as a double vessel. The development of vessels in fresh corneal cicatrices is illustrated in Figs. 1 and 2.

A patient 45 years old had had a severe ulcus serpens on his right eye, which was finally brought to a standstill after cauterization and paracentesis. The causal injury took place June 4th, the beginning of cicatrization was fixed with certainty on July 2d. On Sept. 5th an extensive new formation of vessels was visible in the large leucoma (Fig. 1). The scar from the paracentesis can be seen below as a curved line. The leucoma involves almost the entire cornea with the exception of a small marginal portion. It is penetrated by superficial vessels at the margin and in the central part, partly by the deeper vessels springing from these. To the right a twig can be seen that from the first enters in the deeper layers of the cornea, the clear margin of which is penetrated by many deep loops of vessels, only a few of which reach the leucoma. In the latter are 20 or 25 glomerulus-like vessels usually in direct connection with the larger stems, all with two rather large vessels which may be looked upon as afferent and efferent, though this distinction is not authorized by anatomically visible differences. The portions of the leucoma about the glomeruli differ in no way from the other parts. Two months

To Illustrate Dr. A. Brueckner's Article on "Clinical Studies of the Vessels of the Cornea."

Fig. 6

Fig. 7

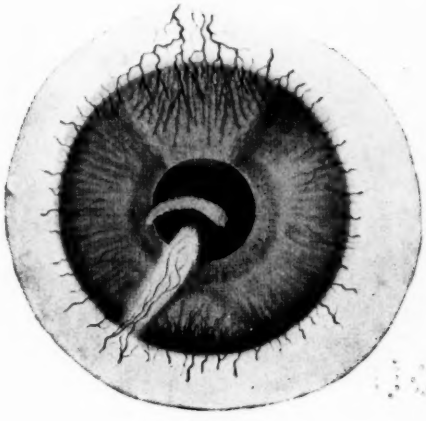

Fig. 8

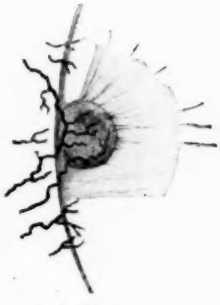

Fig. 9

later the picture had changed (Fig. 2). Little of the former arrangement of the glomeruli remained; they appeared unevenly looser, the individual loop of a glomerulus formed a long arch so that the scar appeared much more clearly vascularized, and the non-vascular parts were much smaller. The process had occupied four months from the beginning of the healing. Unfortunately the patient did not return again.

I observed a similar glomerulus-like new formation of vessels in the following case of a typical parenchymatous keratitis.

A woman 30 years of age had suffered from parenchymatous keratitis of the left eye two years before, which had resulted in perforation and incarceration of the iris. The right eye showed in the centre of the cornea after a preliminary diffuse infiltration a yellowish area which became vascularized from its deep portion very late, after the margin had been penetrated by numerous lamellar vessels and had cleared up. In the middle of this central yellowish spot was a glomerulus of closely connected vessels which was surrounded by an infiltrated, non-vascular area.

A certain similarity was also present in a vascular formation in the leucomatous cornea of a boy 16 years old who had suffered from a lime burn eight years before. The entire cornea was cicatricial and showed two small infiltrated spots near the centre, due to a trichiasis caused by a cicatricial contraction of the conjunctiva of the upper lid. The cornea was extensively vascularized by superficial vessels, single branches of which passed into the deeper layers of the tissue. In the lower portion of the cornea were very numerous minute loops of vessels at regular distances from each other. Under a very high magnifying power the individual loops could be resolved into little vessels passing vertically into the cornea and into afferent and efferent stems. They continued into deeply placed larger vascular stems (Fig. 3). It is worthy of note that this vascular development took place in the region of the cornea affected by the improperly directed ciliæ, but whether the latter was the cause or not I cannot say.

It is certain that in the cases just described the cicatricial structure of the cornea played an important part in the origin of this peculiar form of vessel, for it plainly affords a marked resistance to the outgrowth of vessels. To the same cause

also is to be ascribed the very slow development of the loops in the first case (Figs. 1 and 2). To be sure the rapidity of the development of vessels in the cornea varies greatly. In eczematous processes the development of single vessels can be demonstrated sometimes after a few days, while in infectious ulcers it often begins after several weeks.

3. *Retrogressive metamorphosis of the corneal vessels.*

a. Visible movement of the blood. With regard to the processes that may be clinically ascertained in the retrogression of the newly formed corneal vessels I refer to the works of Friedenwald, Reimar, Augstein, and Best, with whose interpretation of the visible movement of the blood I agree.

It is easily ascertained with the aid of Zeiss's loupe that there is no visible movement of the blood in the corneal vessels at the acme of the inflammatory symptoms. With the retrogression of the symptoms a rapid flowing may be seen in certain veins, which gradually spreads to other vessels and at the same time becomes slower. The segmentation of the blood columns, the slow forward movement of the blood cylinders, has been fully described by Augstein. That this retardation of the movement of the blood runs parallel with the decrease of the inflammation can be well observed in cases that have been treated with jequiritol. Before the treatment is begun, the vessels, so far as they are visible, are only very slightly filled and show a dripping movement. Frequently the movement of the segmented blood column is very slow. As soon as the action of the jequiritol begins, the fulness of the vessels and the swiftness of the movement of the blood increases. As the irritation increases, the movement of the blood gradually becomes less visible until at the height of the jequiritol ophthalmia it is nowhere perceptible. With the gradual decrease of the inflammatory symptoms this phenomenon gradually reappears. If it does not reappear it can be induced to do so by the instillation of adrenalin. From these facts it may be concluded that the appearance of visible movement of the blood in the corneal vessels announces the passing away of the inflammation. It may be remarked that it is not always easy to perceive this phe-

To Illustrate Dr. A. Brueckner's Article on "Clinical Studies of the Vessels of the Cornea."

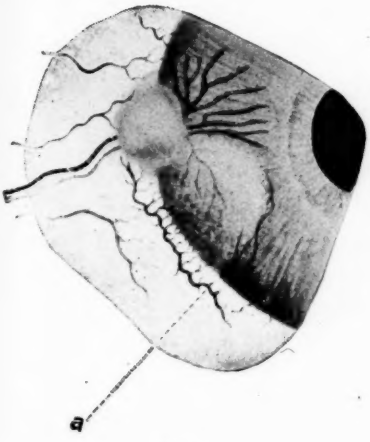

Fig. 10

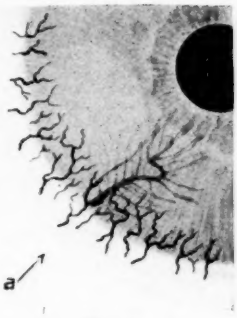

Fig. 10 a

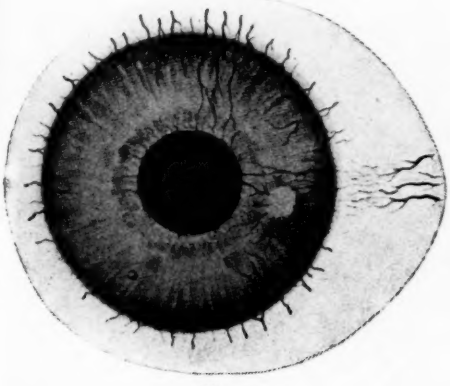

Fig. 11

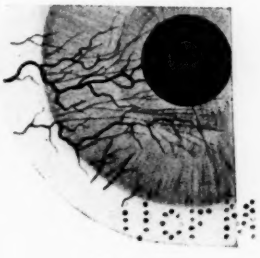

Fig. 12

nomenon, especially when the current is still rapid. Fig. 4 seeks to present the appearance of corneal vessels with segmentation of the blood columns.

The length of the separate stages, from rapid visible movement of the blood to a slower movement, then to segmentation, and finally to almost complete standstill of the blood columns, depends in the first place on the rapidity of the retrogression of the inflammatory symptoms. With the jequiritol treatment these stages require a few days and a marked change often can take place within twelve hours.

The direction of the visible current is centrifugally from the centre of the cornea; it is probably in the veins that the phenomenon is first of all to be seen. Occasionally a centripetal current is present, as in the above mentioned case (Fig. 4). In the large vessels passing across the cornea a current could be seen going from left to right over its entire breadth. Sometimes in the marginal loops of the superficial network which have encroached upon the cornea during inflammation a centripetal current can be perceived in one branch of the loop during the retrogression of the inflammation. It then appears in the connecting portion and then in the other branch, in which the current is centrifugal. Sometimes in one and the same segment of a vessel the current moves first in one direction and then in the other. This is particularly the case when the course of the vessel is not radial. I saw this in an 18-year-old girl who had a small old macula near the limbus of her right cornea with a little encroachment of the marginal network. The short vessels running radially were a fairly large distance apart and were connected by twigs running nearly concentrically to the limbus. In one of the latter the movement of the blood could be seen to be sometimes in one, sometimes in the other direction. Sometimes there were several oscillations of the blood elements which would then be thrown suddenly into one of the supplying vessels when a somewhat stronger impulse sent them in one or the other direction.

More important than this observation seemed to me to be the circumstance that I could detect clearly in these oscillations of the blood column a dependence upon the pulse beat, as the rhythm agreed exactly with that of the pulse in the

radial artery. Hence I may assert that in many cases a continuance of the pulse wave into these little vessels, which closely resemble capillaries, can be seen during life. I have also found a rhythmic movement that accorded with the radial pulse in other cases of marked dripping movement, but yet there is no doubt that movements of the blood columns are more or less arhythmic. At any rate pulsations can be observed in the larger arteries, as could be seen clearly in the case represented by Fig. 13. A girl 12 years old had a band-like ulceration in the upper part of the cornea filled by superficial vessels. Paracentrally below there was a fresh, fairly deep ulcer. Superficial lamellar vessels extended to the latter both from the right and the left. With improvement of the ulcerative process and decrease of the symptoms of irritation the dripping movement became visible in the afferent branches, and in one a rhythmic movement clearly dependent on the pulse beat could be seen. It was much more rapid than the movement in the corresponding veins and therefore more difficult to demonstrate. At the place of transition from the arterial to the venous part of the loop the movement of the blood could not be seen because it was too rapid. I have seen a similar case in a woman 24 years old. Whether this transmission of the pulse into the smallest arteries was due to aortic insufficiency or not I cannot say.

b. Obliteration of the vessels. It has been ascertained by Hirschberg that the deep vessels in interstitial keratitis never completely disappear, but the opinion is widespread that the superficial subepithelial vessels undergo involution. Recently Augstein has pointed out that in almost all corneal opacities single vessels at least may be found by means of the loupe. It seems questionable to me whether an extensive obliteration of the superficial vessels really takes place, for very frequently before the commencement of jequiritol treatment there are almost no vessels in cases in which there is a complete absence of irritation, but within 1 or 2 days after the beginning of the treatment vessels of such length and thickness are present that one may be inclined to the theory that the vessels are old ones which have been not completely obliterated though rendered impermeable to the blood stream.

To Illustrate Dr. A. Brueckner's Article on "Clinical Studies of the Vessels of the Cornea."

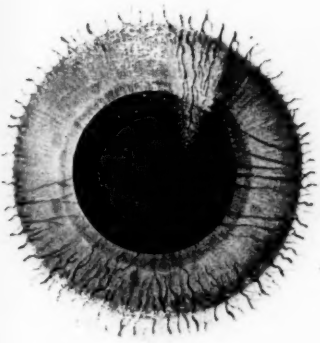

Fig. 13

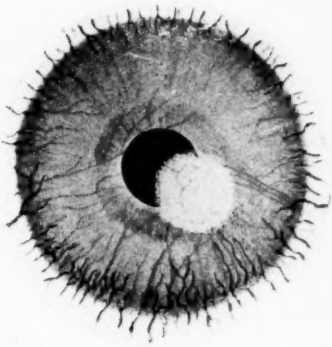

Fig. 14

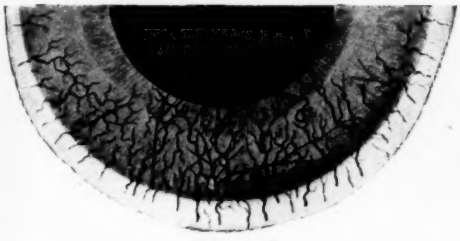

Fig. 15

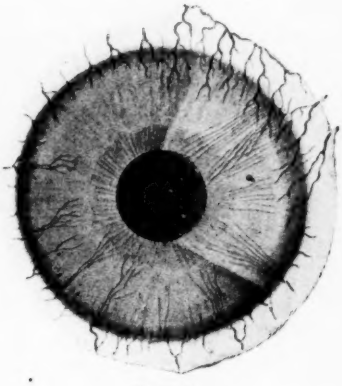

Fig. 16

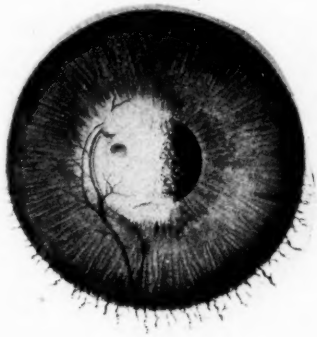

Fig. 17

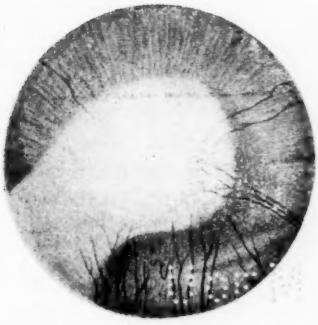

Fig. 18

Physiological Signification of the Corneal Vessels.

It seems to me that there is not yet perfect agreement concerning the physiological signification of the newly formed corneal vessels. Fuchs distinguishes the superficial vessels, which are reparative, from the deep ones in interstitial keratitis, which belong to the clinical picture of the inflammation. Such a distinction does not seem to me justified, for in parenchymatous keratitis we see the advancing development of vessels accompanied by an increasing clearing up of the previously opaque corneal tissue. We must ascribe a nutrient and consequently a reparative function to the vessels in the cornea in all cases. The vascularization in deep ulcerative processes doubtless aids in providing the reparative material for the filling of the defect.

It may be that before cleansing the ulcer the vessels have the task of carrying certain protective materials to the focus of inflammation, materials which could not be carried in sufficient quantity by the lymph current alone. Possibly the extension of vessels into apparently healthy corneal tissue in the neighborhood of the diseased portion may be explained in this manner. Augstein has already pointed out that it is difficult to understand the reason for the penetration of deep lamellar vessels into the clear cornea in iritis. Perhaps this process has a prophylactic signification. In the same way one is tempted to explain the vessel formation in many fresh eczematous processes when they are characterized as such by a fanlike radiation of superficial lamellar vessels into the clear, apparently healthy cornea about an efflorescence lying in or near the limbus (Figs. 5, 6, 7, 9, 10). But such an explanation cannot be given as the true cause, which must always be sought in an irritation that may vary in the individual cases. In deep ulcers it is perhaps of a histo-mechanic nature, but in fresh inflammations it is more frequently abnormal products of metabolism which give a stimulus to proliferation of the vessels on the margin of the cornea. The development of vessels in the larger ulcers of the cornea may be explained thus, especially when they are not placed centrally. The largest vessels come from the parts of the limbus nearest the margin of the ulcer, while

from the more distant portions of the limbus the vessels are smaller or wanting. This is particularly the case with the lamellar vessels. Probably a diffusion of pathological products of metabolism in the neighborhood is responsible for the projection of lamellar vessels into the clear cornea in recent eczematous processes. As to the origin of the vessels in iritis, which extend mainly or exclusively from below into the deepest layers of the cornea, an irritant action of the deposits on the posterior surface may be supposed, but this is purely hypothesis. We know just as little why the development of vessels differs so extraordinarily in different cases of one and the same disease of apparently equal severity. In inflammations of exogenous origin the different virulence of the infecting germs must play a part and more or less must be ascribed to individual predisposition.

II.—Special Portion.

Augstein has already shown that the deep lamellar vessels are not pathognomonic for interstitial keratitis, as thought by Hirschberg. He was able to demonstrate in certain cases which presented a dense network of deep vessels, such as are met with after an interstitial keratitis, that this disease had not preceded. The same author found lamellar vessels in various depths of the cornea in other diseases, though very sparingly. The following observations are confirmatory of and complementary to Augstein's views.

1. Superficial lamellar vessels.

In relatively fresh cases of eczematous affections I have repeatedly seen vessels arranged in the form of fans or feathers which penetrated beneath the marginal infiltrate or ulcer into the transparent area of the cornea. They seem to me to be characteristic of this disease but are not to be observed in all cases. The individual trunks are always from a loop of afferent and efferent vessels.

Fig. 9 was taken from a woman 25 years old who had had an inflammation of her right eye for 14 days. There was a little marginal ulcer which quickly cleared up, together with some very small central infiltrates and deposits on the pos-

To Illustrate Dr. A. Brueckner's Article on "Clinical Studies of the Vessels of the Cornea."

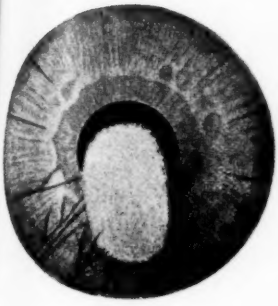

Fig. 19

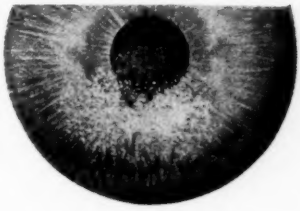

Fig. 20

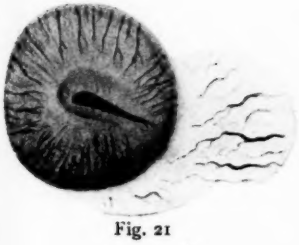

Fig. 21

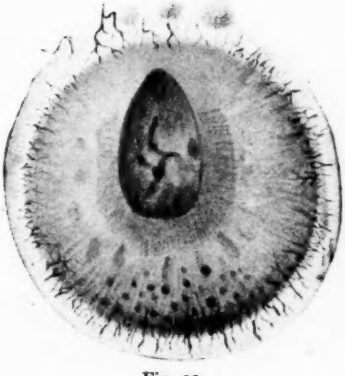

Fig. 22

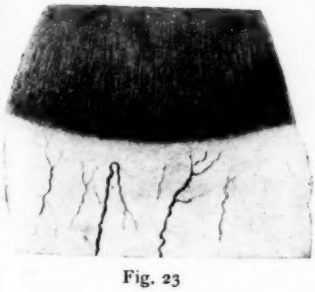

Fig. 23

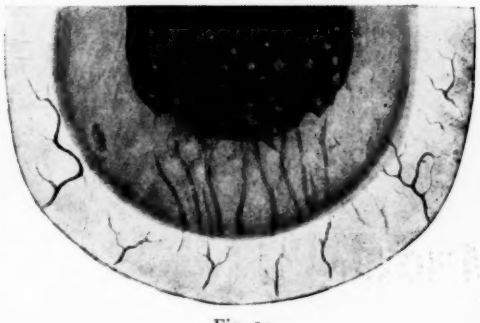

Fig. 24

terior surface. At the beginning of the observation single superficial vessels were already visible proliferating into the base of the ulcer, but in addition there were tuftlike delicate loops of vessels penetrating below the ulcer from 1 to *2mm* into the clear cornea. Below these tufts were similar stems.

That the little marginal ulcer rather than the little central infiltrate was the cause of the development of the lamellar vessels in this case seems to be shown by the following case in which the central corneal affection was wanting.

Fig. 10 represents the marginal portion of the cornea of a woman 22 years old who had a small fresh phlyctenule above an older macula. A lamellar vessel wound about the lower border of the latter (*a*) while a tuft of deep vessels extended beneath the phlyctenule.

Fig. 10*a* shows an eczematous marginal ulcer in a woman 32 years old who had had multiple marginal efflorescences for the first time two months before and at this time was suffering from a recurrence. Near the ulcer, partly encroaching upon it, was a feather-shaped convolution of lamellar vessels (*a*).

Such a tuftlike branching of the vessels may be observed not only in marginal eczematous efflorescences, but also in central infiltrates. In a 6-year-old girl there was an infiltrate about as large as the head of a pin to which ran both subepithelial and superficial vessels from the episcleral network that surrounded the infiltrate with tuftlike stems radiating about it. (Fig. 5.) A similar stellate arrangement about a fresh infiltrate in the cornea of a woman 22 years old is shown in Fig. 6. A similar arrangement of lamellar vessels can sometimes be seen with vascular bands, but they show no stellate appearance because they proceed not only from the margin, but also from the entire course of the band, which gives them an arrangement more resembling an ear of corn or an umbel. (Fig. 7, from a girl 16 years old.)

All of these lamellar vessels had a relatively superficial position. The same is true of the following cases, in which, however, the middle layers of the cornea were occasionally involved in the vascularization.

Figure 8 represents the cornea of a girl 15 years old who had a recurrent eczematous keratitis. Besides a little vascular

band and a central crescentic infiltrate a nearly semicircular area was to be seen above and below through which ran superficial lamellar vascular loops arranged in a tuftlike manner. There were also single short lamellar stems near the limbus.

If the pathological process has existed in the cornea for a longer time this regular arrangement of the vessels may be lost. They then often extend over a large area of the cornea (Figs. 11–13). Fig. 11 was taken from a boy 14 years old who had a fresh infiltrate near old corneal opacities, which do not give the correct impression in the drawing. There were radially arranged vessels, but in addition there were extensive lamellar stems that ran through a great extent of the cornea and could be followed into the superficial marginal network. The conditions were similar in a woman 31 years old (Fig. 12) in whom a fresh process was present as well as older spots. In another case (Fig. 13, a 12-year-old boy) lamellar stems were present together with superficial subepithelial vessels which accompanied a little vascular band. The development of the lamellar vessels was very extensive in the case of a 12-year-old boy (Fig. 14), in whom eczematous keratitis had recurred many times.

Although in the preceding cases the vessels were not too numerous, cases are met with in which the vessels are densely packed together. Thus in a child with severe eczematous keratitis I could see below the margin of a dense superficial pannus lamellar vessels running so close together that even under the loupe the portion of the cornea affected looked like a red cloth. A differentiation of the condition from retrogressing parenchymatous keratitis could not be made.

Single relatively superficial lamellar vessels are to be met with in other corneal diseases than eczematous processes. Thus I saw single short deep loops in a case of trachoma with extensive pannus. In an extensive lime burn of the cornea, after the fresh inflammatory symptoms had passed away, several lamellar vessels could be seen together with a rich superficial vascularization. Deep vessels may also appear after injuries from foreign bodies. Two bits of copper were driven into the cornea near its margin and were not completely removed. Two weeks later two short lamellar loops of vessels

To Illustrate Dr. A. Brueckner's Article on "Clinical Studies of the Vessels of the Cornea."

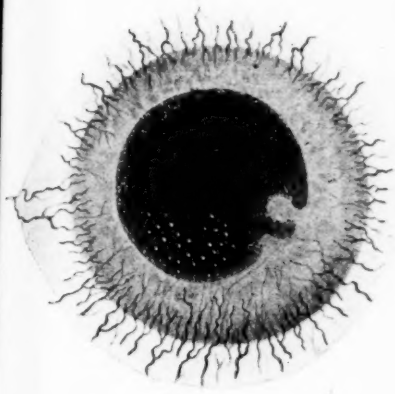

Fig. 25

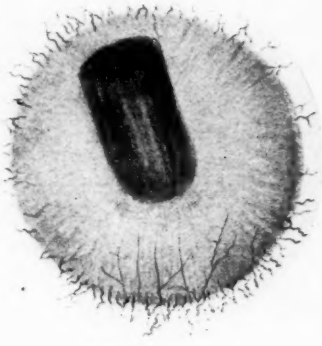

Fig. 26

Fig. 27

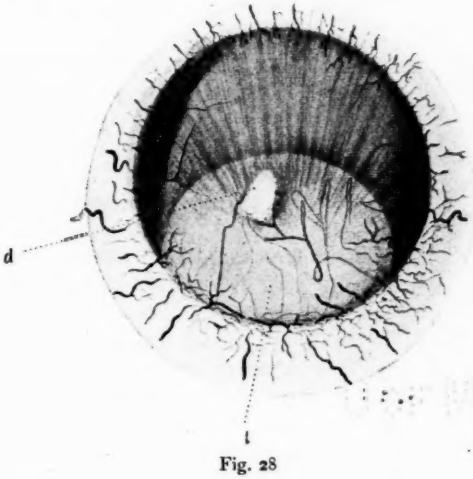

Fig. 28

UCLA

were present, extending to the foreign particles which were healed in and apparently causing no reaction. Superficial vessels were completely absent. Fig. 15 shows the lower half of the cornea of a woman 20 years old who had suffered from facial paralysis on that side from birth. A keratitis e lagophthalmo had developed which had caused an extensive formation of superficial vessels. In addition single lamellar stems were present, some arranged in a radiating manner, others running a course nearly concentric with the margin of the cornea.

In the catarrhal ulcer, as in eczematous keratitis, a superficial, subepithelial formation of vessels is the rule, but occasionally lamellar, relatively superficial vessels are present. Thus I saw in a typical catarrhal ulcer in a girl 20 years old a superficial lamellar vessel which anastomosed at its apex with the subepithelial network. In Fig. 16 is shown the left cornea of a woman 55 years old who had an extensive ulcer involving the central part of the cornea. Together with an encroachment of the superficial marginal network numerous deep birch-broom-like vessels could be seen to extend into the cornea.

2. *Lamellar vessels in the middle layers of the cornea.*

The vessels met with in interstitial keratitis may be taken as the type of those running in the middle layers of the cornea. The vascularization in these cases is not exclusively due to the lamellar vessels, but to the superficial vessels as well, *i. e.*, to branches arising from the superficial marginal network (Fig. 4). The short lamellar loops of vessels present with smaller central ulcers are also usually in the middle layers of the cornea. Whenever a bacteriological diagnosis was made I tried to trace back the genesis of the vessels and find a connection between them and the bacterium, but without success. It has already been shown by other authors that ulcers with large loss of substance frequently heal with the development of deep and particularly large vessels. These vessels differentiate themselves by their dissimilar large calibre from the much more delicate lamellar stems of which we have been speaking. They lie in the middle, sometimes in most pos-

terior layers of the cornea. In Fig. 17 such a healing ulcer is to be seen, with a thinner artery and a thicker vein which do not run parallel throughout their course. In addition there are several smaller lamellar stems which do not reach the ulcer. As the ulcer had cicatrized when the patient came under treatment I could not determine what micro-organisms had caused it.

3. *Deep lamellar vessels.*

In ulcus serpens it is the rule that when the development of vessels begins the deep stems are the ones which have to do with the vascularization. These run in the region between the middle and posterior thirds of the thickness of the cornea. Much more rarely subepithelial and superficial vessels are present.

Fig. 18 shows the cornea of a woman 28 years old who had a pneumococcus ulcer which recovered after repeated cauterizations and paracentesis of the anterior chamber. The drawing was made about 3½ months after the commencement of the sickness. The arrangement of the vessels in the posterior layers of the cornea can be seen beautifully. Superficial vessels were completely wanting.

While in this case the formation of vessels began rather late, about four weeks after the beginning of the disease, deep lamellar stems were present in another case only a few days after the injury. At the time of her admission, three days after the injury, the 38-year-old patient had a pneumococcus ulcer about 3 or 4*mm* in diameter. There were three deep short vascular loops to be seen below and two to the temporal side. Later this vessel formation increased very much. When the ulcer, which had to be cauterized and finally perforated, had got well after five weeks it presented the picture shown in Figure 19. The very early development of vessels had not the power to influence the course favorably.

In contrast with this rapid development of vessels was that met with in another, much less malignant, case of ulcus serpens, in which single short, deep lamellar loops of vessels appeared in the lower and lateral parts of the cornea eight weeks after the injury. These vessels reached nearly to the ulcer which was central and had a diameter of 4 or 5*mm*.

These observations seem to indicate a dependence of the degree of vascular development upon the virulence of the bacterium and the consequent malignancy of the ulcer.

The vessels described by Augstein as present in iridocyclitis of exogenous and endogenous origin occupy the same position in the posterior layers of the cornea, and it may be that in ulcus serpens, in which the anterior portion of the uvea is always involved, the origin of the deep lamellar vessels may be ascribed in part to the iridocyclitis.

Fig. 20 shows the lower half of the cornea of the right eye of a patient 28 years old who had a perforating wound of the cornea and a traumatic cataract caused by a piece of glass. A severe iridocyclitis with hypopyon followed, in spite of which the eye was saved. The picture was drawn 4½ weeks after the injury. Deep loops of vessels came from below, of which only the thicker, venous branches are shown. The white specks indicate the deposits on the posterior surface of the cornea.

After operations followed by an iridocyclitis a vascularization of the cornea may take place. Fig. 21 shows the behavior of the corneal vessels in a three-year-old child in whom infection took place after the extraction of a congenital luxated lens which had caused glaucoma. The picture was taken almost two months after the operation. Below and to the right is the cicatrix of the operation to which the iris is adherent. The cornea shows deep vessels throughout its circumference.

As a transition to the cases of endogenous iritis a case may be mentioned here in which after bilateral extraction of lamellar cataract deposits on the posterior surface of the cornea appeared simultaneously in the two eyes although an interval of eight days had intervened between the two operations. The cause of the iritis could not be determined because the prolapse of iris shown in Fig. 22 was unilateral while the inflammation was bilateral. It healed smoothly and must be regarded as the manifestation of a general infection in a locus minoris resistentiæ. In this case it was marked that the deep vessels in the lower part of the cornea were considerably longer than those that came from the upper and lateral parts of the periphery of the cornea. It is possible that the

vessels appeared first in the lower part of the cornea (compare with Fig. 20). This localization of the deep vessels in the lower portion of the cornea must at any rate be considered as typical in most cases of endogenous iritis.

Fig. 23 shows, greatly enlarged, the vessels running in the deepest layers of the cornea in a woman 23 years old who had an iritis of uncertain origin. The inflammation was possibly tuberculous, for there were isolated nodules in the iris and a large gelatinous deposit in the angle of the anterior chamber. The drawing was completed about 10 weeks after the beginning of the disease when the symptoms were retrogressing.

Fig. 24 shows the appearance of deep vessels in a woman 38 years old who had suffered for two years from a chronic, probably tuberculous, iritis. The vessels extend much farther into the cornea and are much thicker than in the preceding case, yet they are confined to the lower part of the periphery of the cornea.

If the iritis has existed a long time there may be a development of vessels from the entire circumference of the cornea, but those coming from below are longer than those coming from the other parts. Thus in the case depicted in Fig. 25, a woman 25 years old with almost positive tuberculous iridocyclitis, after the disease had lasted several months the lamellar vessels coming from below were the longest of all, while the others were shorter in proportion as their origin was higher. In this case I was able to follow the starting of the vessels successively, first in the lower, then in the lateral, and finally in the upper part of the cornea. There were coarse deposits on the posterior surface which decreased in number upward, and posterior synechiæ. Similar conditions were present in the case depicted in Fig. 26, a woman 40 years old who had had iridectomy performed on account of secondary glaucoma. The etiology of the iritis was uncertain. The gray reflex in the pupil indicates the complicating cataract. That the lower margin of the cornea is the place of predilection for the development of the deep vessels in iritis is confirmed by a case of syphilitic iritis in a man 19 years old. In this case there were vessels in the lower part of the cornea alone, which extended pretty far toward the centre, but originated only from the lower portion of the circumference. Unfortunately

no picture was taken of this case, but it presented a certain similarity to the condition shown in Fig. 24.

The presence of deep vessels is relatively rare in iritis and by no means the rule. I have been able to discover nothing positive as to their origin. They are to be met with in cases of iritis of varying etiology and severity. It has already been suggested that there may be a dependence of the development of the vessels upon the deposits on the posterior surface of the cornea, which would go to explain their exclusive, or at least primary, appearance in the lower part of the corneal periphery. But this hypothesis needs much confirmation because vessels are present in cases that present few and small deposits, while on the other hand they may be absent in spite of large deposits.

Finally I will mention a case, depicted in Fig. 28, met with in a man 55 years old who said that he had formerly been myopic. Detachment of the retina had appeared spontaneously in both eyes, blinding the left and reducing the vision of the right to uncertain perception of light. In the right eye there was a shrunken cataract, while in the left the lens (l) was dislocated downward and adherent with the upper part of its anterior surface to the posterior surface of the cornea. Only the upper part of the iris could be seen. In the central part of the cornea was a vesicular-appearing focus of degeneration (*d*). In the region of the cornea to which the lens was adherent there were elongated loops of vessels running in the deepest layers of the cornea and in addition separate short stems in the upper part of the margin. It must be supposed that the development of vessels is to be ascribed to the contact between the cornea and the lens, for there was no trace of a pre-existing iritis.

In recapitulation the following points may be given in partial confirmation of older writers.

It has not yet been ascertained why in one and the same pathological process in the cornea vessels develop in one case and not in another. Evidently the condition of irritation at the particular time plays an essential part, as well as in the varying rapidity with which the vessels are formed.

The appearance of visible movement of the blood in the newly formed corneal vessels indicates the retrogression of the condition of irritation.

Deep lamellar vessels may be present in the most various diseases of the cornea aside from interstitial keratitis. They occupy a more superficial position in eczematous diseases and in catarrhal ulcers, while in ulcus serpens they are found, if present, in the deeper and deepest layers of the cornea.

In fresh eczematous efflorescences characteristic finger or feather-shaped lamellar vessels are sometimes met with which extend partially into the clear cornea.

A similar ingrowth of lamellar vessels into the clear corneal tissue is sometimes to be met with in iridocyclitis of exogenous or endogenous origin. In the latter case their preponderating or exclusive appearance in the lower periphery of the cornea is to be considered typical. They always run in the deepest layers of the cornea.

STUDIES IN THE PATHOLOGY OF VITREOUS FIBRILS.

By Prof. RICHARD GREEFF, Berlin.

Translated from the German Edition, Vol. liii., No. 2, by Dr. Percy Fridenberg, New York.

NO part of the eye has been so little studied, in a pathologic-anatomic sense, as the vitreous. A study of the publications of the work in this field gives us the impression of results which do not agree or which cannot stand critical analysis. This may depend in part on the technical difficulties met with in the histologic examination of the material in question, the vitreous being, as is well known, so deficient in albumin that it shrinks in most hardening fluids. It is of the utmost importance to go back to the old method of examining fresh specimens.

For some years I have been engaged in a somewhat closer study of the pathology of the vitreous. The results of these investigations, which are in part at variance with accepted views, may be of interest. A survey of the characteristics of the normal vitreous will serve as a preface.

1. *Normal Properties.* The vitreous is not a gelatinous exudate, but a tissue, with a light but firm texture.

The presence of structureless membranes forming a sort of network or honeycomb in the vitreous was formerly assumed by all. Hannover popularized the hypothesis of an orange type for the human vitreous, and of an onion-peel type for that of mammals. This idea has been abandoned, and the work of Bowman, Ciaccio, Retzius, and H. Virchow has led to a general acceptance of fibres or fibrils as structural elements of the vitreous.

The framework of this body consists, then, of a network

(H. Virchow) or tangle (Retzius) of smooth, round fibres. An apparent granulation is to be considered as an artefact. At the intersections (nodes) of three or more threads, there are no knots and no variation in the thickness of the threads; no cells are to be seen here, and in fact the fibres do not seem to have developed from cells. In some cases a number of fibrils meet in a common point, and have a star-shape in consequence. The tangle of fibres is not of uniform density, as they radiate particularly from the region of the ora serrata in the form of long tufts like horse-tails (Retzius) which spread out in the vitreous. In imbedded specimens these tufts have a somewhat curly shape, probably due to the hardening process. The surface of the vitreous is particularly rich in fibres which unite under the hyaloid membrane to form the membrana limitans anterior. Here the meshes are long and narrow, giving the impression of a striated tissue. Toward the interior the meshes become so small that in many places they would not hold a red blood-cell, simulating a molecular structure. The hyaloid canal is surrounded by an area in which the network is denser. The central portion of the vitreous contains very few fibrils and is therefore more fluid. At the points of greater density the fibres may be gathered in lamellæ which directly under the hyaloid membrane appear concentrically arranged. In this way the idea of a series of compartments or chambers in the vitreous becomes intelligible. In fact, their existence forces itself upon us, although we recognize the former conception of a loculated structure like that of the orange to have been exaggerated.

The vitreous is very scantily supplied with cells. That they actually occur is admitted by most authors, but as yet but little light has been thrown on the question of their extent and possible significance. Leucocytes, which may be found anywhere, also occur in the vitreous. These are aliens and are possibly to be looked upon as the product of a natural reaction to special and local conditions within the vitreous. Ordinary polynuclear leucocytes are not uncommon. They may be found wandering about in the periphery, while the central portions are for the greater part entirely free of cells. Under the hyaloid membrane, on the other hand, we regularly find large flattened cells, generally star-shaped, and "ly-

ing scattered at rather decided but not at all regular intervals" (Retzius). Ciaccio assumes that they are in direct contact with the membrane, and calls them "cellulæ subhyaloideæ." According to Beauregard they are scanty in the posterior portions and become more numerous toward the zonula. The cells have long processes and one or more nuclei. Vacuoles are occasionally seen in the protoplasm. Schwalbe also believes these cells to be leucocytes, while later authors are inclined by the uniform character and constant presence of the cells to consider them as fixed. These flat, star-shaped elements would impress us as typical connective-tissue cells, did we not know that leucocytes also have a marked tendency to adhere to surfaces and send out cell-processes along them. Beauregard calls these cells "elements embryoplastiques," and claims that they multiply by division. I cannot accede to this view. The cells undoubtedly increase in numbers under pathologic conditions, but I have never been able to detect karyokinetic figures, and am persuaded that the increase is due to immigration. I am becoming more and more sceptical as to the presence of fixed cells at all. It is certainly more probable that only migratory cells occur in the vitreous.

Besides these *solid constituents* (fibrils and cells) the vitreous contains a *fluid*. Pressed through a filter, the vitreous yields about 98% of the latter. This fluid is very much like the aqueous, being limpid, perfectly clear, of alkaline reaction, and of like specific gravity (1.0089 according to Giacosa). It shows traces of albumin, sugar, urea, and paralactic acid in about the same amounts as the fluid of the anterior chamber.

It is to be noted that the albumin-content of the vitreous increases very markedly not only under pathologic conditions but in the normal eye as well, post mortem, a circumstance which has led to more than one incorrect observation. The aqueous humor and the fluid of the vitreous are most similar in composition to the cerebro-spinal fluid. The scanty residue on the filter consists of hyaloid membrane, fibrillary framework, and cells. The histologic examination of the structure of the vitreous meets with great difficulties, as the vitreous on account of its large water-content shrinks markedly

in all hardening fluids. This contraction invariably takes place in the intact eye in such a way that the vitreous becomes detached from the retina all the way round at the back of the eye, and often appears only as a remnant just behind the lens; it does not adhere at this point, however, the only attachment being to the ora serrata or to the region directly in front.

2. *Fluidification.* Fluidification of the vitreous is a secondary process, occurring when the tissues of the eye which nourish it have been damaged. We agree entirely with the statement of Cirincione that "the importance of the ciliary body as a matrix for the vitreous is amply confirmed by the destructive processes consequent upon lesions of this region." With this structure in a normal condition, the vitreous has the ability, as Zinn has shown, of taking up water and regaining its former bulk after a loss of substance.

We have seen above that the fibrillary network is not of equal density in all parts of the normal vitreous, but that the centre, particularly, is poor in fibres and consequently is more fluid. This becomes more marked in the course of years. Even in middle age, the framework formed by the fibrils is less dense than in youth in consequence of a sort of disintegration, while the most peripheral portions usually appear more fibrillary. This dissolution is often limited to certain areas, so that large cavities form. Retzius says in his great work on the vitreous: "In adult life the tissue of the vitreous undergoes a sort of degeneration, disappearing in part and giving place to accumulations of fluid. These changes may occasionally take place in early life, but are almost the rule later on. The dissolution consists essentially in a rarefication of the framework." In very old but normal eyes, the vitreous may be quite fluid; in horses, this is the rule (H. Virchow). According to Retzius, the senile fluidification begins in the region directly behind the lens, the meshes becoming wider and wider and finally disappearing. Iwanoff found fluidification in the rear portion of the globe accompanied by fatty degeneration of the cells and disappearance of the framework.

Fluidification of the vitreous may accordingly be general or limited. Circumscribed fluidification has been particularly

noted in senility, with the development of irregular cavities. Weiss describes such cavities in the posterior portion of the vitreous in myopia. Carl Theodor observed a series of communicating crevices, which he took to be preformed lymph-channels. Fluidification of the vitreous occurs in the course of various diseases in which the nutrition of the vitreous is affected. It is the rule in chronic cyclitis and occlusion of the ciliary processes. Any morbid change in the pars ciliaris retinæ is accompanied by changes in the vitreous. The fibrillary groundwork of the vitreous seems first to suffer a disturbance of nutrition and dissolves. For a long time fluid continues to be secreted in sufficient quantity, although chemically changed, to prevent any marked loss of tension. If we cut open a globe after irido-cyclitis with adhesions has run its course, the completely fluid, yellowish or greenish, opalescent vitreous immediately runs out. At a later stage, the fluid is secreted in insufficient quantities; tension is decidedly reduced, and the globe feels soft, as if fallen in. The chemical composition of the vitreous fluid is soon and easily affected by pathologic processes. The normal vitreous contains so little albumin that it often shrinks to a small mass in hardening fluids. We have noted above that this takes place soon after death, and that the vitreous is then found to be somewhat richer in albumin. So that a globe taken fresh from the living subject acts somewhat differently from cadaveric material, being more difficult to harden. This is still more noticeable in pathologic conditions, particularly those affecting the ciliary body; the vitreous not only becoming fluid, but showing a greater albumin-content. This is very striking in a hardened globe. The vitreous no longer shrinks, but fills out the interior of the eye completely. I do not believe that the dissolution of the framework is sufficient to account for the increase in albumin. We must assume that some morbid change has taken place in the epithelial cells lining the orbiculus ciliaris and supplying the vitreous, so that albuminoid substances are allowed to pass through from the blood which were formerly held back. The process is quite like that seen in the aqueous, which, as I have shown, also becomes albuminoid and coagulable after detachment of the ciliary epithelium. In such cases various

pictures are presented by the vitreous, which may solidify uniformly, like a boiled egg, and without any shrinkage whatsoever. The corresponding specimen would show, when fresh, a completely fluid vitreous, of green or yellow hue, and quite clear. In other cases, particularly in the presence of a recent, acute inflammation, fibrinogen ferments gain access to the vitreous, and we find the latter filled with a delicate network of fibrin composed of threads varying in thickness from fairly marked strands to almost invisible fibrils (just as it appears in the anterior chamber, in the subarachnoid space, in the retina between the supporting fibres of Mueller, and, finally, in membranous conjunctivitis). This is evidently the condition which is spoken of as *fibrillary degeneration of the vitreous*. We must, however, bear in mind that the framework of the vitreous had disappeared intra vitam, and that the jelly had become quite fluid. The delicate coagulum was formed in the hardening fluid.

3. *Fibrillary Degeneration of the Vitreous.* According to Leber and Nordenson this morbid change is the important factor in retinal detachment which is preceded by a change in the structure of the vitreous, consisting of an increase in density of the normal fibrillary network. Changed in this way, the vitreous has a macroscopic appearance which reminds us of that of another mesodermal tissue, viz., hyaline cartilage, *e. g.*, the costal cartilages, which in old age are also subject to a sort of fibrillary splitting and asbestos-like degeneration. The fibres run in uniformly curving serpentine lines some of which are parallel for long distances, so that wide areas of the vitreous appear folded, while others are arranged in curly tufts of irregular course. Some of these tufts run parallel to the inner surface of the retina in a meridional direction, but the greater part run more or less vertically to it, radiating from the surface of the retina into the vitreous in large tufts. Between the more or less wavy bundles we find cavities filled with a finely granular mass. These granules are to be interpreted as cross-sections of fibrils, or as granular albuminoid coagula. The vitreous which has become thick-fibred, shrinks, and this shrinkage leads to a detachment of the vitreous and later of the retina.

These findings have been repeatedly corroborated, and I,

myself, have seen similar pictures in my specimens, but the interpretation offered by Leber and Nordenson can no longer be accepted. My judgment may be summed up in the sentence: Just as there is no regeneration of the vitreous fibrils, so there is no hypertrophy, thickening, or increase in inflammation or degeneration. So that there is no such thing as fibrillary degeneration of the vitreous, the condition described under that heading in hundreds of publications being evidently something quite different. In the first place, we have normal, preserved fibrils which are crowded together in a small space by the pressure of the detached retina. A like effect is produced by the shrinkage of the normal vitreous in hardening fluids, which generally takes place from behind forward. The fibrils are then crowded together, and are often seen in thick curly tufts streaming out into the vitreous from the ora serrata as described above and depicted in Nordenson's work. This is a perfectly normal condition, and the better the vitreous fibrils are preserved, *i. e.*, the younger and more normal the globe, the better it can be seen, other things, such as the hardening and fixing fluids, being equal. *The only change which the vitreous fibril undergoes is its dissolution.*

We have seen above that this occurs to a certain degree in all eyes as a senile change. It is more noticeable in myopic eyes, as first observed by Arlt in his classical description. Low degrees of near-sight, say 2–3 D., were not accompanied by abnormalities of the vitreous. In artificially hardened as well as in freshly cut highly myopic eyes the residue of the vitreous ran out in flakes or fringes which floated in a serous fluid.

In extensive retinal detachment this change is even greater. The fibrils which at first were merely crowded together, as described above, soon dissolve completely. If we examine a case of funnel-shaped detachment, fresh, what is left of the vitreous is found to consist of a yellowish, mucilaginous mass without the slightest trace of fibril-formation and absolutely free of the retina. This finding definitely does away with the theory of shrinkage of the vitreous as a determining factor in detachment of the retina; a theory which is open to many other objections.

The cicatricial bands, too, which Deutschmann severs in

his operation for the cure of detachment, have never been seen either ophthalmoscopically or anatomically, nor have they been felt with the knife. If this author would examine such cases fresh he would find that, quite to the contrary, the vitreous is very fluid. This picture, of course, changes when the globe has been hardened, as fibril-filaments then develop which are also spoken of as fibrillary degeneration of the vitreous, but which represent something quite different.

In discussing fluidification of the vitreous it was pointed out that under morbid conditions in the eye, particularly in the neighborhood of the ciliary processes, the framework of the vitreous body rapidly liquefies, while the vitreous fluid continues to be secreted for a long time although its chemical composition is greatly changed. An increase in albumin, as was noted above, takes place under inflammatory conditions and may cause fibrin-coagulation which in the hardened specimen is also interpreted as fibrillary degeneration. Intra vitam there would have been a gelatinous fluid which after resorption of the normal mass appears as an albuminous exudate coagulating to a network in the hardening fluid.

4. *Detachment of the Vitreous.* The occurrence of this pathological process was first noted by H. Muller, and Iwanoff, later on, discovered that *posterior detachment* of the vitreous is characteristic for a number of ocular affections. It was found that the vitreous, retaining its gelatinous consistency, became separated from the posterior part of the globe; the space thus formed was filled with a fluid of independent origin, an exudation which was not to be confounded with fluid vitreous, as the rear boundary of the detached vitreous was always sharply defined, and the tissue itself unaltered. If we are to consider the fluid accumulation, not as an exudation, but as pathologically altered vitreous, it should contain vitreous cells. Of 30 cases of vitreous detachment examined by Iwanoff, 11 were due to intraocular changes following injury. Three of the eyes showed marked axial myopia, but were otherwise unaltered. Iwanoff considers his detachment analogous to Arlt's fluidification of the vitreous. He says: "In the space which forms between vitreous and retina, serous exudate accumulates more and more as the staphyloma develops, and detaches the vitreous

farther and farther from the retina. In most of the cases the detachment of the vitreous is a prodroma of the retinal ablation. At the margin of the detachment, the vitreous becomes adherent to the retina, and so finally limits the regionary extent of the detachment. The accumulating fluid can now no longer find room between the vitreous and the retina, and must either tear through and penetrate into the vitreous, or by continued pressure burst the retina and cause its detachment from the choroid." Iwanoff's investigations have been confirmed by O. Becker, Pagenstecher, and Genth, and particularly by Carl Theodor, Leber, and Nordenson. Sections made at points beyond the equator of the lens also show the shrunken vitreous detached in front from the zonula fibres, so that there is an *anterior detachment of the vitreous* as well. "This formation of a post-zonular space (Czermak, H. Virchow) cannot well be ascribed to shrinkage due to hardening, as the crevice is occupied by a mass which has coagulated during the process of hardening." Shrinkage of the vitreous may exert traction on lens, zonula, ciliary processes, and iris. Particularly if the attachment of the anterior surface of the vitreous to the zonula fibres be intimate, the latter with the ciliary processes and the root of the iris may be drawn backward by the contracting mass of the vitreous. This may cause a deepening of the anterior chamber which at times acquires diagnostic significance pointing to vitreous detachment.

The histologic descriptions of Iwanoff and, later, of Nordenson are unusually thorough and conscientious. My own researches confirm them, but I must disagree with their interpretation. The normal vitreous is adherent particularly in front in the region of the ora serrata. When hardened, it contracts from behind forward, the fibrils coming closer together. As the fluid normally present in the vitreous is not rich in albumin, it does not coagulate, and so hardly appears in the hardened specimen. The albumin-content increases markedly, however, under pathologic circumstances, and we accordingly find after hardening that the vitreous tissue in contracting from behind forward has pressed out the altered fluid, which then coagulates in a homogeneous layer, devoid of fibrils, between vitreous and retina. I am quite convinced, that in

most cases the detachment spoken of was an artefact caused by hardening, and have expressed this view as far back as 1902. Continued studies have served only to confirm my opinion. Further corroboration is given by recently published investigations of Elschnig (end of 1904), who carefully examined 17 hardened eyes with myopia of from 2 to 3 dioptries and 5 globes with atypical myopic refraction,—with staphyloma inward, upward, or downward, or coloboma at the nerve-head. In only one eye was there an *apparent* detachment of the vitreous, *i. e.*, a globular mass of shrunken vitreous attached to the lens and ora serrata; the interior of the globe filled with clear fluid holding delicate flocculi in suspension. More thorough examination showed that delicate vitreous-like masses adhered at all points to the retina, and the microscope revealed the presence of the more or less clearly marked limiting membrane in contact with the retina. Carl Theodor has noted similar conditions, but considers the globular mass adhering to the lens after vitreous detachment as evidence of a degenerated vitreous. In the rest of the eyes normal conditions were found as to the topography of the vitreous and its spatial relations.

Elschnig concludes with a statement, which I have also made, that the idea that detachment of the vitreous frequently occurs intra vitam in eyes affected with posterior staphyloma is not supported by the findings, although this interpretation has been generally applied to artefacts due to shrinkage in hardening.

5. *Regeneration.* As far as the framework and cells of the vitreous are concerned, I am convinced that there is no such thing as actual regeneration. In this I agree with Cirincione, in particular.

The vitreous attains complete development before the end of fœtal life, and appears as a well-formed structure with close meshes. As the vitreous grows larger, the fibrils grow longer, without however increasing in number, and the meshes become successively wider, taking up a larger quantity of aqueous fluid. It is to this circumstance and not to any multiplication or increase in the framework that the growth of the eyeball is essentially due. In the fully developed vitreous, destruction of the framework such as accompanies

fluidification in hemorrhage, high myopia, choroiditis, etc., is not followed by regeneration. The same remarks apply to the fixed cells of the vitreous. I have never been able to find karyokinetic figures either in the histologic examination of material taken from typical clinical cases or in experiments on rabbits. In this point I find myself in accord with Cirincione and Haemers, and, according to a verbal communication, with Krueckmann (as opposed to the statements of other authors).

Wounds of the vitreous may be experimentally inflicted and studied in two ways: first, by extracting the lens and, after the section has healed, puncturing or incising the vitreous from in front through cornea and pupil; or, secondly, by penetrating the interior of the globe through sclera, choroid, and retina at the equator.

Only the fluid of the vitreous is renewed, and this, according to most authors is secreted by the pars ciliaris retinæ, a single layer of high cylindrical cells, gradually becoming lower, which form a continuation of the retina and cover the pigment layer from the ora serrata on as far as the orbiculus ciliaris and the ciliary processes. In this way we can understand how a moderate loss of vitreous is soon made good and the normal tension of the globe restored. It cannot be denied that most of the published investigations come to positive results in regard to regeneration. The studies of Carl Theodor and of Haemers are particularly to be mentioned as unusually complete and careful, although I have been obliged in this difficult question to come to entirely different conclusions. The former author found regeneration of the vitreous four times in a series of 20 eyes with detachment of the vitreous. It appears in two forms: 1, as a thin layer or lamella covering the inner surface of the limiting membrane over a stretch of several millimetres; or 2, more circumscribed as irregular, gelatinous prominences on the limitans.

Authors who accept a regeneration of the vitreous as a fact, look upon the retina as a matrix from which the cells enter the vitreous.

"It is sometimes possible to observe the transit of such cells from the retina into the vitreous even in quite normal eyes if numbers of microscopic sections are made through retina

and vitreous" (Carl Theodor). That the cells required for the regeneration of the vitreous are derived from the retina was, however, most clearly seen in a case of total staphyloma. "This eye showed, besides detachment of the vitreous, important changes in the retina in which the regenerative process in the vitreous could be followed step by step. The layers of the retina were pretty well preserved on the whole, except that the nerve-fibre layer showed at some points, particularly next to the vessels, an inhibition with serous exudate and a shredding out of individual fibres. The limiting membrane was bowed into the interior of the globe by the bases of Mueller's supporting fibres, which were swollen and lengthened. The layer of rods and cones was swollen only in the detached portions of the retina, but normal at all other points. The inner surface of the limitans was studded with small, flat protuberances, which on microscopic examination proved to be cell-groups. These cell accumulations caused a wavy separation of the fibre-layer from the limitans, the spaces thus produced being filled with cells, and on the other side there was a similar circumscribed accumulation of cells. The double contour of the limitans formed a sharp dividing line between these two cell groups. At some points a cell or two appeared to be incarcerated in the limitans itself, so that, considering the other pathologic findings, a migration of cells to the inner side seems placed beyond doubt. Although the accumulations just described showed only closely packed cells, a fibrillary intercellular substance could clearly be seen at other points of the cell conglomerations. This evidently constituted a later stage of development, presenting two forms of internal arrangement. In one, the intercellular substance had already become changed into a tenacious, transparent, lamellar mass, which, except for a somewhat increased cell-content, could not be distinguished from the ordinary vitreous."

Of course, the objection may be raised that the bits of vitreous seen on the retina were remnants of the original, now detached, vitreous; a point which the author himself brings up and attempts to invalidate.

The first section of Haemers's (1903) study deals with the development of the vitreous.

This author comes to the conclusion that the vitreous is of ectodermal origin, and reports numerous experiments in regeneration which lead to the view that vitreous is renewed from the retinal connective tissue, *i. e.*, the neuroglia.

After partial loss of vitreous the residue collects at various points, but most noticeably at the ciliary processes and at the ora serrata, where the fibrillary structure could plainly be seen. The normal cells have entirely disappeared. In their place there soon appear numerous very small cells with a large nucleus and very little protoplasm: lymphocytes. Haemers observed no mitoses which would suggest a regeneration by karyokinesis. The fibrils stand in intimate relation to the sustentacular tissue of the retina (Mueller's supporting fibres) from which they seem to have originated, and can be followed into the deepest layers.

The newly-formed elements consist of

1. A granular network.
2. Cystoid, globular cells.

After partial loss of vitreous, increased secretion soon equalizes the hypotony of the globe. In a day or two the tension is again normal and the fundus oculi appears unchanged except in those cases in which the retina had become detached. This increased transudation to compensate for lowered tension is only provisional, preceding true regeneration. After a time a network appears composed of numerous exceedingly fine fibrils which intertwine without joining. They make their appearance clearly only at the surface of the retina upon which they end in a broadened base, or they can be seen coming up from the deeper retinal layers beside the supporting fibres of Mueller.

The second variety of regeneration consists in the formation of globular cells, which are found at the equator of the eye and at the ciliary region in more or less dense accumulations. They are scarcer in the posterior part of the eye, and may at times be seen welling out of the surface of the retina. They have nothing to do with the blood-vessels.

It seems to me that the embryologic data, especially together with other circumstances, are opposed to the conclusions of these authors. It would certainly be remarkable if the vitreous were to be regenerated from any tissues but

those from which it develops embryologically, *i. e.*, the orbiculus ciliaris or the ora serrata, according to the consensus of most authors.

Conclusions.

The vitreous fibril, once it has attained full growth, is unchangeable; incapable of division, reproduction, or after-growth; so that there is no regeneration of the vitreous, although there may be fresh secretion of fluid. There is no fibrillary degeneration, which presupposes division and proliferation of fibrils. A distinction must, of course, be made as to "organization" of the vitreous, in which neuroglia, connective tissue, and blood-vessels proliferate into the interior. The only change the fibrils undergo is their dissolution in morbid processes in the region of the ciliary body and orbiculus ciliaris.

PATHOGENESIS OF THE TRAUMATIC AFFECTIONS OF THE ORBIT. (EMPHYSEMA ORBITÆ; HÆMORRHAGIA RETROBULBARIS; ENOPHTHALMOS TRAUMATICUS.)

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Translated from the German Edition, Vol. lii., No. 3, by Dr. Matthias Lanckton Foster.

(*With one figure in the text.*)

THE orbit is of importance not only for the protection and preservation of the eyeball and its adnexa, but also on account of its direct connection or close proximity to the cranial cavity and the accessory sinuses. The diseases of the orbit belong primarily to ophthalmic science, but the general surgeon and rhinologist are also interested, because they often play a determining part in difficulties of differential diagnosis. The possibility of an orbital complication during the course of accessory-sinus disease is not unimportant to the rhinologist, while in injuries of the skull and of its contents the orbital symptoms are of special interest to the surgeon. The great importance of an undisturbed functional capacity of the contents of the skull and the compression of vital organs confined in so narrow a space demand a priori that all symptoms coming from the part under consideration should be most carefully considered in order to obtain points for diagnosis and prognosis. Several years ago Bergmann remarked the semeiotic importance of conjunctival, palpebral, and orbital extravasations from the surgical standpoint. Speaking as a rhinologist Onodi pointed out many dark places in our understanding of the ocular symptoms which accompany accessory-sinus disease and demanded for success-

ful work the association of ophthalmologist and rhinologist. To the ophthalmologist those processes are of special interest which change the normal nutrition and functional conditions of the eyeball. Injuries of the orbit are directly or indirectly of etiological importance in the retrobulbar tumors, Basedow's disease and phlegmon of the orbit. Most of the lesions which are not associated with fracture of the skull and the consequent menacing symptoms are of moderate severity. The position of the orbit, partly surrounded by hollow cavities, is such and the variety of its contents is so great that the clinical pictures which result from injuries are of great variety, and one and the same injury may produce different forms of disease according to the portion of the contents, or of the walls, of the orbit affected.

Let it be supposed that through violence the lamina papyracea of the ethmoid or the floor of the frontal sinus is pierced so that during forced expiration air enters the orbit, or that a blood-vessel is wounded either by fracture of one of the walls of the orbit or more directly, so that its contents escape into the orbit, the closure of the latter in front always remaining intact, the most prominent symptom produced by such a diminution of space by air or blood will be an exophthalmos. If the displacing elements are spread evenly, the eye will be protruded directly forward in the direction of the orbital axis to a degree proportionate to the quantity of those elements, and as a necessary consequence of the increased tension of the extrinsic muscles the motility of the eye will be interfered with to a degree which will vary with each individual. In addition to these two symptoms there may be organic changes in the eye and disturbances of the vision from an associated injury, or from pressure upon, tension, or tearing of the circulatory, motor, and nervous apparatus, clinical pictures which will vary from case to case because the irregularity of the acting force produces a certain inconstancy of symptoms.

This slight sketch indicates how varied are the traumatic diseases of the orbit. They are rare in ophthalmic practice. Berlin calculated that they form 5% of all diseases of the orbit, which in turn form 0.19% of all ophthalmic diseases. Comparatively few articles on this subject have been pub-

lished and the following cases may prove a welcome contribution, particularly as a simple treatment is presented which has hitherto been but little used.

I.—Emphysema Orbitæ.

A man 58 years old was attacked on a country road three days before coming to the clinic and received so severe a blow from a fist on the outer portion of the left eye that everything became black before him and he lay for several minutes unconscious. When he reached the nearest village his eye was protruding, the same as it was when he came to the clinic. During the previous year his eyes had been examined and a central scotoma with amblyopia from tobacco and alcohol diagnosticated.

Vision. Right, fingers at *2m*, left at *3m*, the same as in the preceding year, but the test of the vision of the left eye was possible only when the patient raised the upper lid with his finger. There was a marked protrusion of the left eye, ptosis, and a narrowing of the palpebral fissure to about *5mm*. The upper lid could not be actively raised nor the palpebral fissure closed. The movements of the eye were restricted in all directions, upward they were totally lost, downward they were very slight, inward and outward they were better. The left pupil was *7mm* in diameter, the right *4mm*; both reacted sluggishly. The same central scotoma was present for white and colors as formerly with paleness and excavation of the temporal side of each papilla, but not the least difference between the ophthalmoscopic conditions of the two eyes. There were no palpebral or conjunctival ecchymoses, except that at the outer canthus there was a slight, bluish discoloration of the skin. There was no swelling or distention of the lid with air and the upper lid was stretched tightly over the eyeball. The swelling of the retrobulbar tissues which produced the exophthalmos felt uniformly elastic and yielded to pressure only slightly. Such pressure caused pain. Pressure of the globe against the inner wall of the orbit was very painful, as was also the attempt to palpate the latter. No crepitus could be detected with certainty, but the resonance on percussion with the tips of the fingers on the lids showed the presence of air in the cellular tissue of the orbit. The needle of an aspirating syringe was introduced deeply into the orbit through the lower conjunctival fornix and a quantity of air in bubbles of bloody froth, but no fluid blood, was withdrawn. The eye sank back immediately into its normal position.

In the afternoon the exophthalmos had recurred to the same degree in spite of a firm pressure bandage, and the patient stated of his own accord that on blowing his nose he had noticed that the eye protruded again and the lids had become tense. This had been accompanied by great pain. On the following day the exophthalmos was unchanged. An incision 1½*cm* long was then made in the outer third of the lower lid, through which air escaped in great reddish bubbles, while the eye immediately sank back. A tampon was placed in the incision and a firm pressure bandage was applied. About midday, after a painful blowing of the nose, there was a slight recurrence of the exophthalmos and on the following day the eye was about 5*mm* too prominent. During this day the exophthalmos completely disappeared and the eye went back to its normal position. The movements of the lids and of the eyeball were free and unrestricted, the pupils equal in size and condition otherwise as before the injury. Vision, fingers at from 4 to 5*m*. After remaining four days in the clinic free from pain the patient was discharged wearing a pressure bandage.

The cause of the emphysema which produced the exophthalmos in this case is demonstrated almost with the certainty of an experiment. The classical symptoms which indicate a purely orbital emphysema through fracture of the lamina papyracea are so unmistakable that a critical study of them is unnecessary. Heerfordt's article (*Arch. f. Ophth.*, Bd. 58, Heft 1) may be referred to for an account of the cases hitherto recorded, so it is not needed that they should be described here. This author, on the basis of his collation of the published cases, two cases of his own experiments on the cadaver, divided the changes about the eye caused by the entrance of air into four groups, the purely orbital, the orbito-palpebral, the palpebral, and the conjunctival emphysema. The first and last are very rare; the second form is the most frequently met with.

This classification is based on anatomical grounds. When the power of resistance of the septum orbitale has been overcome by the pressure exerted by the air behind it, the membrane ruptures and the air escapes into the loose palpebral connective tissue. This mechanism is perfectly clear and the conception is not to be undervalued in the differentiation

between orbital and palpebral emphysema. But the differentiation between orbito-palpebral and palpebral emphysema may often be impossible unless made arbitrarily. Heerfordt himself says that exophthalmos may be absent in the former. But if this one differential point is wanting, what is there to authorize the theory of an orbito-palpebral emphysema? There remains only the characteristic swelling of the lids, a true palpebral emphysema. Certainly the origin of the latter as given by Heerfordt is the more frequent and the older theory of a perforating injury of the lachrymal bone or of the lachrymal sac must be exceptional.

But there is another possibility to be borne in mind, which comes from an accurate anatomical study of the layer of fascia which closes the orbit in front, a subject which is treated of exhaustively by Merkel in his text-book. He describes as a constant anatomical condition three spaces in the septum orbitale which serve for the passage of vessels and nerves. Two of them lie at the upper margin of the orbit between the bone and the fascia to serve for the passage of the supraorbital and frontal nerves and vessels. The third lies over the internal palpebral ligament and permits the anastomosis between the orbital and facial vascular systems. In the lower lid the tarso-orbital membrane is more delicate, the fibres are not so close together, so that the closure of the orbit is not so firm as above. Merkel points out that the septum orbitale in connection with the fasciæ of the muscles, forms an almost complete closure, and that tumors and effusions which lie outside of the muscular funnel must appear between the eyelid and the margin of the orbit when they advance from the latter.

This anatomical postulate is thus confirmed by clinical experience. It is also to be considered that the ecchymoses which are of such diagnostic importance in fractures of the base of the skull come about in this way. But what may be assumed for a fluid material may be assumed with still greater probability for air. Heerfordt mentions in the description of his experiments that in some cases just before the rupture of the orbito-palpebral membrane the latter forms a protrusion filled with air, usually just above and below the internal palpebral ligament. If this statement is

compared with the picture on page 85 of Merkel's work, the thought will be suggested that this has to do with the above-mentioned third space for the passage of anastomosing vessels. At any rate the escaping air finds a more favorable opportunity to spread in the loose tissue of the lids than in the contracted unyielding cavity of the orbit.

Pure orbital emphysema is of the greatest rarity and its occurrence, or rather the non-occurrence of the palpebral or orbito-palpebral form, is to be explained, according to Heerfordt, by the occasional ability of the tarso-orbital fascia to resist the pressure from within the orbit. A closure by retrobulbar tissues of the places where the orbital septum is perforated might be possible, or, in rare cases, such a valvular arrangement of these places might be conceivable that a complete closure would be produced by the increased intraorbital pressure.

Several years ago Fuchs stated that emphysema is almost always found beneath the loose skin of the lids and that the purely orbital form is extremely rare. He considered that the most frequent cause was a fracture of the lamina papyracea of the ethmoid bone produced by the impact upon it of the eyeball acting as a medium for the traumatism. Such an impact may result in a scleral rupture, or in a fracture of the orbital wall, accordingly as the power of resistance of the eyeball or of the lamina papyracea is the greater. This theory is upheld by Walser's experiments and also by the clinical fact that emphysema and scleral rupture have never been observed together. Fuchs explains the fact that emphysema of the orbit is so seldom met with in great injuries by stating that usually there are solutions of the continuity of the skin through which the air easily escapes. Hence in cases of emphysema the injury is not very severe, the prognosis is good, and in only exceptional cases is the eye seriously injured.

Operative intervention is indicated only in cases of such high degree as the one here reported. Puncture in this case was very efficient in relieving the eyeball, and was not only of great therapeutic value, but also of very great importance in establishing the differential diagnosis.

II.—Retrobulbar Hemorrhage.

A boy 12 years old was struck on the left side of his forehead, four days before his appearance at the clinic, by a stone weighing about two pounds, thrown by another boy. The blow knocked him down and rendered him unconscious for several minutes. There was bleeding from the wound in his forehead, but not from his nose, ear, or mouth. When he returned home there was a great protrusion of his eyeball.

On the left side of the forehead there was a gaping lacerated wound about *7mm* long, starting at the outer end of the inner third of the superciliary arch and running obliquely upward and inward. There was an exophthalmos of 8 or *10mm*, the upper lid was tense and immovable over the protruding eyeball. The palpebral fissure was about *7mm* wide, rather wider than the right, but the pupil, which reacted normally, was not visible and the lid had to be lifted passively in order to test the vision. The motility of the eye was restricted in all directions, least downward. The lids were black and blue and there were several ecchymoses of the conjunctiva. The vision of the right eye was $\frac{6}{6}$, that of the left $\frac{6}{12}$, not improved by glasses. Both eyes were emmetropic and ophthalmoscopic conditions were perfectly normal.

Emphysema of the orbit was improbable because of the gaping wound in the brow and of the sugillation of the lids and conjunctiva, while a retrobulbar abscess was excluded by the slight signs of irritation in the eye itself and the normal temperature. Thus by exclusion was made a diagnosis of retrobulbar hemorrhage, perhaps from direct injury to the supraorbital artery or vein, perhaps from fracture of one of the bones of the orbit.

The wound was uninfected and was sewed up on the following day. The exophthalmos remained unchanged, but the vision had fallen to $\frac{6}{30}$. In order to hasten the slow absorption of the hæmatoma and prevent further injury to the eye, an aspirating needle was introduced through the outer part of the upper fornix deeply into the orbit and about *15ccm* of brown, partially clotted blood was withdrawn. During this procedure the eye sank backward and finally became replaced in its normal position, where it remained. After three days the patient was discharged cured with a vision of $\frac{6}{6}$.

Through the kindness of his physician I learn that up to the present time, eight months after the receipt of the injury, the recovery has remained perfect. The vision is $\frac{6}{6}$, there is no difference in the size of the pupils, and

the position of the eye is normal without a trace of exophthalmos. There is a slight ptosis of the upper lid.

Such cases are mentioned much more rarely in ophthalmic literature than would be expected from the frequency of injuries to the head and the consequent opportunities for the production of retrobulbar hemorrhages. This may be due in part to the fact that in certain cases, such as fracture of the base of the skull, the orbital trouble forms only a symptom of a more serious injury. These patients pass into the hands of the general surgeon and the ophthalmologist sees only the less serious cases in which there are no symptoms of general trouble. The first and only monograph on this subject is that by Berlin in Graefe-Saemisch's Handbook, and his description may, with few limitations, be considered correct to-day.

Beside hemorrhages into the orbit as the result of traumatism, cases of so-called spontaneous hemorrhage have been observed. Berlin quotes three in which the persons affected suffered from the hemorrhagic diathesis. Holmes Spicer, Philipson, and Conkey have seen subperiosteal hemorrhages in badly nourished cachectic children. Van Santen and Hirsch observed the occurrence of exophthalmos during stooping due to the rupture of a varix, with local vascular disease. Berlin saw an orbital hemorrhage caused by an unusually severe fit of coughing, Friedenwald and Crawford one due to changes in the vascular walls ascribed to alcohol and nicotin poisoning. Panas considered that an exophthalmos due to hemorrhage from rupture of the vessels was caused in a reflex manner by the stomach through vascular paralysis. Böhm described an attack of retrobulbar hemorrhage in a woman 26 years old during parturition. Shaw observed an exophthalmos from hemorrhage into the orbit in a woman 57 years of age who had not menstruated for 5 years. Finally Morax saw a sudden hemorrhage which was, as he thought, of a venous nature, and produced marked exophthalmos without demonstrable cause.

This brief review can make no pretence to completeness, because I was unable to obtain a number of articles in the original, but an examination and criticism of the preceding

cases is indispensable for the elucidation of the clinical picture. It is plain that our knowledge in regard to the pathogenesis of exophthalmos due to non-traumatic hemorrhage is still very slight and that almost all the published observations correspond to the hypotheses ascribed for their explanation. When due to traumatism there are only two possibilities to be dealt with. The hemorrhage may be due either to a direct wound of the orbital vessels, as in penetrating wounds of various kinds which usually come from in front, or to indirect laceration of vessels in the orbit produced by violence to the bones of the orbit or to some other part of the skull. These orbital hemorrhages, as has already been pointed out, are of great semeiotic importance because almost without exception they form a symptom of fracture of the orbital wall and are usually associated with other serious injuries.

The exceptions—that is, the cases in which orbital hemorrhage has occurred without fracture of the orbital wall—are comprised in Hölder's six observations, published by Berlin, and Morian's last. In all these seven cases there was a severe injury: three jumped from the third story, one fell on a pile of stones, one fell from a scaffold two stories high, one fell into a quarry 80 feet deep, one was struck with the blunt end of a hatchet on the left side of the head. All resulted in death within a week, but the autopsies showed that in none had there been a fracture of the orbit or of the skull. According to Berlin this would go to show that orbital hemorrhages following severe contusions of the skull occur in 91–92% with, and in 8–9% without, fracture of the walls of the orbit.

Here also belong the so-called congestive hemorrhages after compression of the body described by Perthes, Braun, von Recklinghausen, Niemann, and Morax, and briefly collated by Wienecke. These were all cases of severe injuries, contusions, and bruises of the chest or abdomen, in which beside ecchymoses in the skin of the neck, head, and occasionally the arms, the eyeballs protruded in consequence of hemorrhage into the orbit and the vision was more or less disturbed, for the most part temporarily. The following theory has been advanced as an explanation of the occurrence of such hemorrhages. The increased air pressure in

the thorax due to the compression of the body presses upon the large veins and produces a wave of engorgement in the opposite direction to the blood current. The reflux of venous blood stops at the right auricle, as shown by two autopsies. Through this passive congestion of the blood the tension in the smaller veins and capillaries is increased so that they burst unless they find sufficient counter-pressure. Nearly the entire venous system of the head and neck is without valves. There are no valves in the internal jugular vein and only a few insufficient ones in the external. Hence when the pressure is increased there is no hindrance to a backward wave in these vessels. With three exceptions intraocular hemorrhages have not been met with in such cases. The explanation of this may be that the intraocular tension, like the intracranial, prevents the occurrence of hemorrhage, as it prevents an acute stasis in the veins of the body from affecting the retinal veins and capillaries with the suddenness and to the degree that it affects other vascular regions.

Aside from an ischæmia of the papilla in some cases, fundus changes have not been observed in exophthalmos from orbital hemorrhage except by Pagenstecher. The latter gives these particulars. A quarter of an hour after the receipt of the injury, while the exophthalmos was still visibly increasing, the case came under observation. There was a great reduction of vision. Ophthalmoscopically there was a diffuse, porcelain-like discoloration of the retina, most marked about the nerve and macula, the arteries and veins were small and the papilla was almost bloodless. Half an hour later the vessels had become uniformly larger, but the discoloration of the retina remained. Four hours later this had partially retrograded and after a few days only a few traces of it could be seen in the form of whitish spots scattered about in the periphery of the fundus. The eye finally became blind from detachment of the retina. Whether such circulatory changes in the retinal vessels could be found more frequently if the patients came under observation at an early stage of the trouble is conjectural. They might be expected at first in hemorrhages from injuries of the arterial branches of the ophthalmic, but, as has been said, in most cases the ophthalmoscopic condition was negative.

Since the publication of the above cited work of Berlin, and in addition to the observation of Pagenstecher just referred to, cases of orbital hemorrhage due to traumatism have been described by Priestley Smith, de Vincentiis, Friedenwald, Crawford, and Hahn. They all presented the well known clinical picture. Hahn's case was one of the rare observations of hæmophilia in a feeble individual who sustained a deep penetrating wound of the orbit as the result of a fall from a wagon. The hemorrhage was uncontrollable and the eyeball finally protruded as far as the angle of the mouth, while its anterior portion became necrotic. Death occurred in eight days from a high septic fever. The autopsy revealed necrotic, purulent masses of granulations in the orbit, with multiple ecchymoses in the brain.

The prognosis of orbital hemorrhages which come under the observation of the ophthalmologist is relatively good, though much less favorable than that of emphysema. The differential diagnosis between these two conditions is made by the presence of sugillations in the conjunctiva and lids and the absence of resonance on percussion in cases of hemorrhage. In doubtful cases the diagnosis can be determined conclusively by puncture, in the manner employed in the two cases here reported; an operation which is absolutely without danger when properly performed and one which produces an excellent therapeutic result.

The interference with vision and the dilatation of the pupil which existed in this case during the exophthalmos can be explained most simply, in the absence of objective signs, by the pressure exerted on the circulatory and nervous conducting apparatus of the eye by the blood poured out into the retrobulbar space. After the evacuation of the hæmatoma both of these symptoms completely and permanently subsided. No enophthalmos resulted as might perhaps have been expected. The slight ptosis, which remained as the only permanent trace of the previous condition, may have been caused by an injury to the levator.

III.—Enophthalmos Traumaticus.

Since Nieden called attention in 1881 to the clinical picture of traumatic enophthalmos a considerable number of cases

have been reported, but its pathogenesis has been by no means elucidated. On the contrary, almost every new observation has been accompanied by a new hypothesis, each able to explain single symptoms, yet not one competent to explain the development of the entire complex on a single basis. Three years ago Lederer collated the published cases in a tabulated review, criticised the hypotheses which had been advanced, and sought to establish an explanation which would account for its occurrence in all cases. But such an attempt has little prospect of success when one takes into consideration the complicated and peculiar anatomical conditions which pertain to the structure, position, and contents of the orbits, and how the clinical pictures produced by similar injuries must consequently vary. The three cases here reported give evidence of this and a series of similar cases may be readily collated from literature in which the traumatism consisted of a contusion in the orbital region, while the resultant injuries and their pathogenetic explanations were extraordinarily different. Our knowledge in regard to traumatic enophthalmos therefore cannot be considered so conclusive as to render useless the publication of the following characteristic and accurately observed case, particularly as it depicts the clinical picture with great clearness without disturbing accessory symptoms.

A man 46 years of age had received an injury two months previously which had led to a lawsuit and caused him to come to the clinic to obtain an authoritative opinion upon the condition of his eye. A horse had kicked him near the left eye severely enough to knock him down and render him unconscious for several hours. There was no bleeding from his nose or mouth. The swelling of the lids and of the left side of the face was so great that he was able to open his eye only after its subsidence, at the end of a week. He then noticed at once that the "eye lay deeper in the orbit and was smaller than the right." He wanted damages on account of the great pain he had suffered and was still suffering at times in the lower bony margin of the orbit, which had appeared immediately after the injury and was ascribed by his physician to a fracture of the bone. He had not noticed any reduction of vision and had not had diplopia.

With correction of the hypermetropia, +1.50 D., the

vision of each eye was $\frac{6}{6}$ and Nieden 1 could be read with +2.50 D. The left palpebral fissure was 1 or *2mm* narrower than the right and there was a moderate degree of enophthalmos (*3mm*) of the left eye. The upper tarso-orbital fold was deep, but the movements of the lids were normal. The movements of the eye were unrestricted upward, outward, and inward, and were a little greater downward than those of the other eye. On palpation of the two eyes the finger passed more deeply into the left orbit and more of the left eye could be felt than of the right. Prolonged stooping, pressure and compression of the cervical jugular did not alter the condition of enophthalmos. There were no other changes. The pupillary reactions were the same as in the other eye, the fundus was normal. The left pupil and palpebral fissure promptly dilated after the instillation of three drops of a 5% solution of cocaine into the conjunctival sac, which demonstrated that the sympathetic nerve was performing its functions normally and that its participation in the etiology of the clinical picture could be positively excluded.

Near the outer end of the inner third of the lower bony margin of the orbit was a distinctly circumscribed, slightly thickened place, which was tender and was designated by the patient as the site of his pain from the beginning. Prof. Seifert confirmed the opinion that this was a callous on the site of a fracture or infraction. He also was so good as to examine the accessory sinuses and found them intact.

Ten months later an examination revealed the same condition in all respects except that incipient cataract had appeared in both eyes and had produced a fairly uniform reduction of vision.

For a review of the literature on the subject of traumatic enophthalmos I refer to the work of Lederer which has already been mentioned, and I agree with the views and criticisms of this author in regard to the theories which have been advanced. Therefore I shall refrain from any discussion of these points and confine myself to a brief study of two hypotheses which seem to have more claim to correctness than most of the others.

In 1888 Gessner advanced the theory that enophthalmos was produced by the eyeball sinking back mechanically into the orbit in consequence of a cicatricial contraction of the retrobulbar fat and the pressure of the eyelids. To this

theory Beer made the correct objection that it is conceivable that an inflammatory contraction of such nature as to produce the setting back of the eyeball could take place in the brief space of eight to fourteen days, the time which had elapsed between the receipt of the injury and the appearance of the enophthalmos. But his contention is incorrect that such an inflammation cannot result from a crushing blow which penetrates to the bone. A retrobulbar phlegmon, with its very deleterious consequences, is not a necessary accompaniment, as Beer claims when he says: "Gessner speaks of no such condition, but claims that the bones are slightly involved as the result of the traumatism and that clinically such injuries frequently show a tendency to inflammatory reaction and to the transmission of this to the periosteum and to the retrobulbar fat."

Though the first objection is doubtless correct, I do not think Gessner's hypothesis should be absolutely rejected; the possibility of such an inflammatory reaction with subsequent cicatricial contraction of the orbital tissues exists. The theory is favored by the fact that in the great majority of cases a penetrating wound was made by the injuring blunt force, and in many the underlying bones were involved. A scar was absent almost exclusively in the so-called pure, uncomplicated cases of traumatic enophthalmos, *i.e.*, those in which the changed position of the eyeball is the only result of the injury, while the other conditions of the eye, particularly its motility, remain normal. The entire number of such cases are those reported by Nieden, Beer (second case), Denig, Shoemaker, Kilburn, and the one here narrated.

The second theory was advanced by Lederer. He assumed a direct or indirect fracture of the wall of the orbit, with hemorrhage into the orbital tissue which resulted in a cicatricial formation which in turn brought about the retraction of the eyeball. All of the various symptoms which accompany traumatic enophthalmos he ascribed partly to direct simultaneous lesion of individual orbital tissues by the hemorrhage, or by fragments of bone, and partly to the involvement of the adjoining tissue in the cicatrization of the orbital wall. The purely mechanical production of the displacement of the eyeball backward through cicatricial contrac-

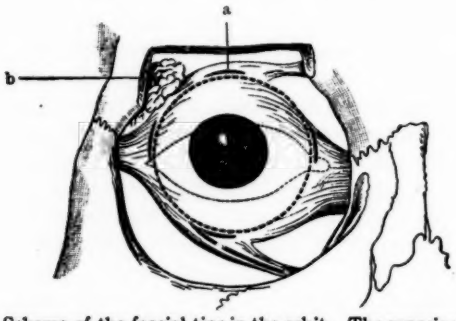
tion is common to both theories, but while Gessner assumed an inflammation of the retrobulbar tissues as the primary cause, Lederer assumed the presence of a retrobulbar hemorrhage.

A fracture of one of the orbital walls is the most frequent cause of hemorrhage into the retrobulbar fat, and the tenability of Lederer's theory depends on the question whether an orbital hemorrhage can produce such a contraction of the orbital fat as to bring about such an alteration in the position of the eyeball. Clinical experience and the study of the reported cases of retrobulbar hemorrhage answer this question in the negative. As Berlin states, the outcome of this trouble is usually a complete restoration of the functions of the eye, and only exceptionally does the absorption of the blood extend over a longer period than three or four weeks. In the literature regarding traumatic enophthalmos there exists almost no unobjectionable observation in which a protrusion of the eye, the cardinal symptom of a retrobulbar hemorrhage, appeared primarily after the injury. From the pathologico-anatomical standpoint, according to Marchand, the transformation of a hemorrhage into a cicatrix cannot be absolutely excluded, but it is rare. Chiari asserts that the possibility of such a cicatrization with a resultant enophthalmos rests principally on analogy to the apoplectic cicatrices in the brain.

If it is granted that these exceptional conditions may occasionally be met with, a point remains to be considered which is of great importance, but was not dealt with by the author of this hypothesis. This is the fact that the onset of the enophthalmos after the receipt of the injury and the time necessary for the formation of a cicatrix are not in corresponding proportion. The objection which renders Gessner's theory untenable is at least equally as good here. A glance through the recorded histories shows that the interval of time which elapsed between the receipt of the injury and the first observation of the enophthalmos varied between two and fourteen days. The statement is frequently made that the altered position of the eye was noticed as soon as the swelling of the lids subsided. Some of these observations doubtless were made within the short space of fourteen days,

but as that statement is not positively made they should not be included. The number of the positive observations is sufficient to establish the fact that enophthalmos has been seen in a shorter time after the traumatism than it would be possible for cicatrization to take place in retrobulbar tissue after a hemorrhage.

From the foregoing it is evident that both Gessner's and Lederer's hypotheses assume processes and conditions as acting in the pathogenesis of traumatic enophthalmos which have an actual basis and may sometimes play an important part, but are insufficient to explain all cases.



Scheme of the fascial tips in the orbit. The superior orbital margin is removed in part, exposing the superior tip a; b is the lachrymal gland. (After Merkel.)

There must be a supporting power which comes into play and may call forth enophthalmos either directly or at least shortly after the traumatism, even though not to the degree usually observed after the lapse of a longer time. I think this power is present and serves to fix the eye in its position.

It is necessary here to consider the anatomical conditions in order to define more accurately the portion of fascia which is of the greatest importance both for the retention of the eyeball in its position and for the physiological function of the ocular muscles. The description given by Merkel, which I have followed in the main, has been confirmed in its principal points by Motais. The accompanying cut is a schematic representation of the anatomical conditions present. It must

be noted first of all that the muscular fasciæ which are dealt with here are in no way identical with the septum orbitale which was mentioned when speaking of emphysema of the orbit. They unite and together form the closed, aponeurotic funnel of the orbit, but the septum has no influence upon the position of the eyeball.

The fascia which envelops the muscles and joins them on the one side with the eye and on the other with the orbital wall is only a delicate membrane over the retrobulbar portion of the muscles, but at their anterior ends it forms dense, connective-tissue bands which divide in a Y-shaped manner at the place where the tendon passes through an opening in Tenon's capsule to be inserted into the eyeball. One leg of the Y blends with Tenon's capsule so as to form a close, firm union with it at the place of passage of the eye muscles, knowledge of which is of importance in operations for strabismus. The other portion thickens into a fairly dense membrane which arches out from the side nearest the orbital wall and is firmly inserted into the periosteum of the inner side of the margin of the orbit. There are three such insertions, one to the temporal side, one to the nasal side, and one below. The one on the nasal side is divided into two unequal portions, the broader one below and the narrower one above. The fasciæ of several muscles blend together. There is no insertion above because the tendon of the levator passes between the eyeball and the bone.

The function of this attachment of fascia, in which Sappey demonstrated smooth muscle fibres, is twofold. If the insertions are cut away from the bone the eyeball will rotate so that on freeing the superior rectus the cornea looks toward the anterior part of the roof of the orbit, an unnatural position. Hence this arrangement of the fascia acts in the first place as a guide to the direction of the movements of the ocular muscles.

A second function is manifested on section of the bands themselves, the eyeball sinks back into the orbit. This is because the portions of fascia attached to the bones of the orbit hold Tenon's capsule in its normal position as a consequence of their close union with it. The lower part of this arrangement of fascia is very firm and is so directed that the

eyeball rests in it as in a sling. For this reason Lockwood has given this connective-tissue band the name of ligamentum suspensorium oculi.

If we apply these anatomical facts to the clinical picture of traumatic enophthalmos, it appears that in the majority of cases at least the enophthalmos cannot occur without a lesion or a loosening of the bands of fascia which have been described. This idea is by no means new. Purtscher has already discussed the mechanical force which can produce the slightest dislocation of the eyeball. More recently Shoemaker has declared the importance of this condition in the pathogenesis of enophthalmos. He thinks that the bands of fascia either may be relaxed through atrophy of the smooth muscle fibres in them as a result of paralysis of the sympathetic, or may be torn by a traumatism.

Against this hypothesis Lederer has raised the objection that such a loosening or injury of the fascia must result in a certain instability of the globe, and refers to the rare cases of so-called intermittent ex- and en-ophthalmos to explain which Vossius years ago assumed a relaxation of these bands of fascia as that which rendered possible the change in the position of the eyeball.

Intermittent ex- and en-ophthalmos is very different from the clinical picture now under consideration. It is a chronic trouble which lasts for several years before the onset of the enophthalmos. The primary cause is the presence of varicosities in the orbital veins. Exophthalmos is produced by the overdistention of the veins, the attachments of the fascia become stretched and loosened, and finally enophthalmos is induced by atrophy of the retrobulbar fat. But it is to be noted that the exophthalmos is produced mechanically and that the eye protrudes not only when the patient is stooping, but that it is driven forward by the distended varices when the patient is in an upright position through the mechanical influence of forced expiration or compression of the jugular vein. In intermittent exophthalmos we have therefore not to deal with an instability of the eyeball in that it is abnormally movable on account of its weight. Among other things, the fact that diplopia, or muscular disturbances, have never been observed, indicates the contrary, as does also the fact

that the change in the position of the eyeball is always in an anterior-posterior direction. The completely undisturbed functional capacity of the motor apparatus which was present in our case must necessarily correspond to a normal, or at least to a not noticeably changed, muscle tonus, and this again could be the power which holds the eyeball in position after the fascia has given way.

On the basis of what has been said, I think the etiology of traumatic enophthalmos can be thus explained. A laceration or rupture of some or all of the bands of fascia is produced by a contusion of the orbital region, almost as a rule with fracture or infraction of the bony margin of the orbit. According to clinical experience, as shown in the second section, if this results in a retrobulbar hemorrhage the latter will usually be absorbed without any further damage to the eye, but in isolated cases in which other retrobulbar portions of the contents of the orbit are involved in the injury further damage may result. As has been stated, it cannot be denied that Gessner's hypothesis contains something of value as a secondary influence in the pathogenesis of traumatic enophthalmos and a cicatricial contraction after inflammatory reaction is conceivable.

These cases may be classified in two categories according to the degree and the manner of production of the enophthalmos. The first class includes those in which the enophthalmos is wholly due to a lesion of the bands of muscle fasciæ. The case reported here belongs to this class. It may be termed an uncomplicated case from the absence of all other changes. Such cases are very rare and present only a slight degree of enophthalmos, the limit of which can with difficulty be expressed numerically because under physiological conditions the individual positions of the eyeballs as regards the mouth of the orbit vary greatly. To the second class belong those cases in which after lesion of the fascia the eye becomes fixed in its position in the way suggested by Lederer or Gessner and usually retracts still farther. These cases are the more frequent in literature and practice, and in almost all, other injuries are associated, particularly of the motor apparatus.

Finally it may be that none of these methods affords a

satisfactory explanation of the clinical picture in isolated cases. In these we can not entirely dispense with the so-called nervous theory which assumes an irritation or paralysis of the sympathetic nerve, though it has met with little sympathy because of the almost complete absence of basal facts.

REPORT OF THE PROCEEDINGS OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

By Mr. C. DEVEREUX MARSHALL.

An ordinary meeting of the Society was held in the Medical Society's Rooms, Chandos Street, Cavendish Square, on Thursday, June 9, 1910, Dr. G. A. Berry, President, in the chair.

Mr. C. Blair showed a case of **extensive subconjunctival rupture of a buphthalmic eye;** recovery; also brother of the above patient—also the subject of buphthalmia, and complete congenital dislocation downwards of lens.

Mr. R. W. Doyne showed a case illustrating a not uncommon degree of word-blindness, guttate iritis, and test lenses for the dark room.

Mr. A. H. Thompson showed a case of **vertical nystagmus,** with movement so fine as to escape notice with the naked eye.

Messrs. E. Treacher Collins and A. C. Hudson exhibited microscopical sections of an eye with unusual yellow patches in the iris shown at the last meeting. The patches consisted of large polygonal cells with small shrunken nuclei and spongy cytoplasm, closely packed in a fine reticulum in which the degenerated chromatophores of the iris could be distinguished. The large cells were thought to be probably degenerated endothelial cells. A flat preparation of the choroid from the same eye showing very extensive colloid body formation was also shown.

Mr. Arnold Lawson showed a case of **scirrhus of the orbit.**

Mr. Lang exhibited a case of **growth under the conjunctiva.**

Dr. Rayner D. Batten showed a case of **Raynaud's**

disease, and Mr. POOLEY (Sheffield) a case of **traumatic condition of the retina.**

Mr. ERNEST CLARKE read a paper on **optic atrophy** following the use of arylarsonates in the treatment of syphilis. The first case was that of a man aged 46, who was given ten injections of 5 grains of soamin every alternate day, and after the last the sight became misty. Several months later he had three further injections of 5 grains. He had vision of barely hand movement in one eye and $\frac{6}{12}$ with scarcely any field in the other. Both disks were atrophied. The next patient, aged 49, had 10 grains of orsudan on alternate days and after the 9th injection the sight began to fail. Both disks became atrophied. He then stated what other writers had found after administration of the drugs atoxyl and soamin, and the number of cases in which optic atrophy had followed was considerable. The condition of the disks in these two patients left little doubt but that the drugs were responsible. Possibly the combination of arsenic, anilin, and syphilis might be particularly destructive to the optic nerves, but the lesson he wished to point out was that great care should be exercised in the administration of arylarsonates; the eyes should be most thoroughly examined before and during the administration of the drug; that disease of the arteries, kidneys, or liver contraindicates its use; that the dose should be carefully regulated according to the body weight and age of the patient, and it was doubtful if it ever was safe in any one over 40 years of age. Colonel Lambkin, who is the great advocate of the treatment, had used it mostly, if not entirely, in young subjects. He concluded by saying that when the profession had fully recognized the limitations of the use of the arylarsonates, it might be that they would prove to be all that could be desired of them, but question would arise as to the expediency of employing them at all for the treatment of syphilis. It was one thing to risk blindness in being treated for a fatal disease like sleeping sickness, and quite another to risk it in the treatment of syphilis, even if in some cases it were more successful than the ordinary and well tried methods.

Mr. N. BISHOP HARMAN read a paper giving details of the **inheritance of congenital cataract** in nine families. Of these

five were of lamellar cataract, one of coralliform cataract, one of discoid cataract, and one of form unknown, as the cataracts had been removed some years ago, and one of posterior polar cataract with microphthalmia. In the families with lamellar cataract several showed an inheritance through several generations and in numerous individuals. In one a marriage of first cousins seemed to accentuate it in their progeny. Another of these families showed grave mental disorder on both sides of several generations; several dying mad on the paternal side, and many suffering from epilepsy on the maternal side. The case of posterior polar cataract with microphthalmia was particularly distressing, for of five children born to an apparently healthy couple, of whom two were twin, the four surviving were all irremediably blind from the defective growth of the eye. It was noted that in several instances those affected with congenital cataract were mentally defective.

Mr. WRAY, discussing Mr. Clarke's paper, pointed out the importance of giving the arylarsonates in accordance with the body weight. Cases of blindness after its administration had been numerous after the substance was given and it occurred in two sets of cases—after sleeping sickness, and after syphilis. 15% of cases of syphilis recovered without the administration of mercury at all. Too little had been heard about the Wassermann reaction in treatment; it was used chiefly in diagnosis. Mr. BREWERTON, the PRESIDENT, Mr. DAWNAY, Mr. DOYNE, Mr. TREACHER COLLINS, Mr. MAYOU, Dr. FARQUHAR BUZZARD, Mr. POOLEY, and Major MOLE discussed the case.

Mr. DOYNE showed some cases among members of his first family of "Family Choroiditis" and pointed out that the spots were exudations beginning in early adult life, but more generally later, increased very much in middle age, when, though the appearance of the lesion was gross, the sight was not proportionately affected, and finally in old age passed into atrophy with very great loss of sight.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE THIRD AND FOURTH QUARTERS OF THE YEAR 1909.

By Dr. G. ABELSDORFF, in Berlin; Prof. ST. BERNHEIMER, in Innsbruck; Dr. O. BRECHT, Prof. R. GREEFF, Prof. C. HORSTMANN, and Dr. R. SCHWEIGGER, in Berlin; with the assistance of Prof. A. ALLING, New Haven; Prof. E. BERGER, Paris; Prof. CIRINCIONE, Genoa; Dr. DALÉN, Stockholm; Prof. HIRSCHMANN, Charcow; Dr. J. JITTA, Amsterdam; Mr. C. DEVEREUX MARSHALL, London; Dr. H. MEYER, Brandenburg; Dr. P. VON MITTLESTÄDT, Metz; Dr. H. SCHULZ, Berlin; Prof. DA GAMA PINTO, Lisbon; and others.

Translated by Dr. MATTHIAS LANCKTON FOSTER.

Sections I.–III. Reviewed by Prof. C. HORSTMANN, Berlin.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

171. MEYERHOF, M. **Some new discoveries of manuscripts of Arabic ophthalmologists.** *Centralb. f. prakt. Augenheilkunde*, xxxiii., 11, p. 321.

172. SOMMER, E. **Professions and the blind.** *Deutscher Blindentag in Dresden*, June, 1909.

173. MUSKENS, L. J., and SNELLEN, W. **Results of an investigation into the causes of blindness among the students in the Institute for the Blind in Amsterdam.** *Nederl. Tydsch. voor Geneeskunde*, 1909, Bd. i., No. 17, Bd. ii., No. 15.

174. HOLT, E. **Physical economics.** *Trans. Amer. Ophthalm. Soc.*, 1909.

175. SCHIRMER, O. **Organic and psychogenous disturbances and their relation to simulation.** *Strassburger med. Zeit.*, 1909, Heft 2.

176. VAN LINT, A. *Ocular Accidents from Electricity.* Brussels, 1909.

177. DUANE, A. **A classification of eye diseases.** *Trans. Amer. Ophthalm Soc.*, 1909.

MEYERHOF (171, **Some new discoveries of manuscripts of Arabic ophthalmologists**) found in the library at Cairo seven

ophthalmological manuscripts which complete Hirschberg's work concerning Arabic ophthalmology.

According to SOMMER (172, **Professions and the blind**) the blind are excluded from the study of medicine and the natural sciences. A blind physician may become scientific in a literary sense, or may be efficient as a masseur, or as a physician for speech defects. As a lawyer he can be only a counsellor or an assistant. In theology the outlook is more favorable, as well as in the various sciences linked with the philosophical faculty.

MUSKENS and SNELLEN (173, **Results of an investigation into the causes of blindness among the students in the Institute for the Blind in Amsterdam**) report 22 clinical histories. Of 98 students in the Institute 20 had optic atrophy determined with certainty to be the result of a healed brain disease. As only children with good understanding are admitted, the percentage is probably higher. In only one case had a palliative trepanation been performed, although too late, and a slight amount of vision had been saved. In the majority of cases the primary disease had been "encephalo-meningitis serosa migrans." An operation should be performed if the choked disk does not retrogress in a few weeks and if it exceeds 2 D.

B. P. VISSER.

HOLT'S (174, **Physical economics**) paper demonstrates a mathematical formula for determining the earning ability of a person—either to rate him or obtain his economic value and to ascertain values for damages to certain parts of his body in a manner equitable to all concerned. ALLING.

In addition to the psychogenous disturbances, in which an erroneous condition of the consciousness calls forth bad vision, there exists, according to SCHIRMER (175, **Organic and psychogenous disturbances and their relation to simulation**), a group of visual disturbances which stands between those of organic and those of psychic origin. To this group belongs the amblyopia from non-use in congenital cataract. In these cases the operation for cataract must be undertaken early in life for preservation of useful vision. In amblyopia of a squinting eye, that eye must be used for seeing by excluding the good eye with atropin, concave glasses, or a bandage. The amaurosis after lid spasm belongs not in the psycho-optic

but in a transcortical centre. The fatigue of the visual field as well as the bilateral amblyopia and amaurosis in neurasthenia and hysteria belong to another group of psychogenous visual disturbances.

VAN LINT (176, **Ocular accidents from electricity**) speaks first of lightning, then of the influence of high-tension currents, electric burns, the accidents from wireless electricity, the electric light, and finally the accidents met with in the use of electricity for therapeutic purposes.

DUANE (177, **A classification of eye diseases**) points out the importance of a uniform medical nomenclature. The classification which he offers is based upon that in Nomenclature of Diseases proposed by the College of Physicians in London. The list is most complete, and the author rightly advocates its adoption in compiling hospital and dispensary reports, in making mortality and morbidity returns, and by physicians generally in reporting cases in medical journals, books, and discussions. The advantages in all physicians using the same terms with the same meaning are evident.

ALLING.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND TREATMENT.

178. ROEMER, PAUL. *Experimental and Clinical Foundations for the Serum Treatment of the Pneumococcus Infection of the Human Cornea (Ulcus Serpens).* Wiesbaden. J. F. Bergmann, 1909.

179. ROEMER, PAUL. **The quantitative proportions of the antibodies in the cornea in passive immunization; a contribution to the serum treatment in ophthalmology.** *Deutsche med. Wochenschr.*, 1909, No. 45.

180. TRUBIN, A. E. **The importance of certain kinds of mould fungi in infectious diseases of the eye.** *Russk. Wratsch.*, 1909, No. 31.

181. WLADISCHENSKY, A. P. **The influence of oxygen upon cultures of the staphylococcus pyogenes aureus, and the oxygen treatment of purulent keratitis in rabbits.** *Westn. ophthalm.*, 1909, No. 11.

182. SCHNEIDER, RUDOLF. *Experimental Studies Concerning the Value of "Leucine" in the Treatment of Infectious Inflammations of the Conjunctiva.* Leipsic, W. Engelmann, 1909.

183. VEDERAME. **Experimental studies concerning the action of the bile and its salts upon pneumococci.** *Annal. d'ophtalm.*, 1908, No. 12.

184. JAKAHASHI, U. **Studies concerning the influence exerted upon the germ contents of the conjunctival sac by operations on the globe.** *Klin. Monatsbl. f. Augenheilkunde*, xlvii., ii., 6, p. 741.

185. WESSELY, H. **Experiments on the growing eye.** *Muenchener med. Wochenschrift*, 1909, No. 44.

186. STANCULEANO, G., and MIHAIL, D. **The pathology of the ophthalmo reaction.** *Archiv f. Ophthalmologie*, lxxiii., No. 1, p. 70.

187. LEBER, ADOLF. **Serodiagnostic studies in syphilis and tuberculosis.** *Archiv f. Ophthalmologie*, lxxiii., No. 1, p. 1.

188. SCHUMACHER, G. **The serodiagnosis of syphilis in ophthalmology, together with remarks concerning the relations of tuberculosis to syphilis in eye diseases.** *Deutsche med. Wochenschr.*, 1909, No. 44.

189. JACKSON, EDWARD. **Blood pressure and its practical relations to ophthalmology.** *Ophthalmic Record*, October, 1909.

190. STARGARDT, A. **Disturbances of dark adaptation.** *Archiv f. Ophthalmologie*, lxxiii., No. 1, p. 77.

191. BIRCH-HIRSCHFELD, A. **The changes in the anterior segment of the eye after frequent irradiation with short-waved light.** *Archiv f. Ophthalmologie*, lxxi., No. 3, p. 570.

192. DAVIS, A. E. **The treatment of the eye when the globe is infected, with the object of preventing panophthalmitis.** *Trans. Amer. Ophthalm. Soc.*, 1909.

193. ELSCHNIG and ULBRICH. **Etiology and prophylaxis of the post-operative inflammations of the eye.** *Arch. f. Ophthalm.*, lxxii., No. 3, p. 393.

194. PFALZ, G. **Treatment and after-treatment of wounds and burns of the eye.** *Zeitschr. f. Augenheilkunde*, xxii., No. 6, p. 492.

195. ELSNER, H. H. **Clinical and experimental studies concerning the cause of the symptoms of intoxication after the use of atropin in children suffering from diseases of the eye.** *Ibid.*, xxii., No. 5, pp. 387 and 507.

196. POSEY, W. C., and SAUTER, A. C. **An ophthalmological phase of the dangers of consanguineous marriages.** *Ophthalmic Record*, Sept., 1909.

197. FRAZIER, C. H. **The surgical aspect of cerebral decompression.** *Jour. Amer. Med. Assoc.*, Sept. 11, 1909.

ROEMER (178, **Experimental and clinical foundations for the serum treatment of the pneumococcus infection of the human cornea**) gives the results of his experience with the serum treatment of the ulcus corneæ serpens and speaks particularly of the variability in virulence of the pneumococci. He considers at length the bearing of aggressine, opsonins, and the theory of bacteriotropism upon the serum treatment of ulcus serpens. The entire problem is much more complicated than it has hitherto seemed to be.

According to ROEMER (179, **The quantitative proportions of the antibodies in the cornea in passive immunization; a contribution to the serum treatment in ophthalmology**) the ophthalmologist must determine whether he will not obtain

better results in the treatment of ulcus serpens if he uses larger quantities of the serum than he has hitherto been accustomed to. He has occasionally met with stocks of pneumococci in ulcus serpens which are not influenced by the pneumococcus serum alone. Aside from the general immunization of the entire organism, the serum may be applied locally to the eye in the form of powder, or a dilution may be injected subconjunctivally or into the cornea.

TRUBIN (180, **The importance of certain kinds of mould fungi in infectious diseases of the eye**) studied the aspergillus fumigatus, aspergillus nidulans, aspergillus ochraceus, mucor corymbifer, and mucor rhizopodiformis. Inoculations of the aspergillus fumigatus into the vitreous of rabbits produced in two or three days violent symptoms of irritation and inflammation of all the membranes, which lasted five or six days and then resulted in atrophy of the eyeball. At the end of the second week there was a purulent infiltration of the vitreous and uvea with detachment of the retina. In various places in the vitreous numerous fungous threads were to be found surrounded by pus corpuscles. After five weeks masses of giant cells were to be found in the vitreous and greatly changed filaments of aspergillus in the protoplasm. The life of the fungus in the rabbit's vitreous does not exceed sixteen days; after this time attempts at cultivation give no result. The aspergillus ochraceus produces less intense, but similar symptoms. The symptoms induced by the aspergillus nidulans are still less intense. Infection of the vitreous with mucor corymbifer and mucor rhizopodiformis causes another clinical picture—moderate symptoms of irritation, which disappear almost entirely in three or four days; but the fundus gradually becomes more cloudy and in six or seven days presents the picture of a pseudo-glioma. At this time an exudate is to be found in the eye, in the densest parts of which are filaments in the form of thick threads with leucocytes attached. The same differences in the inflammatory symptoms are to be observed when the infection is introduced into the anterior chamber. HIRSCHMANN.

WLADISCHENSKY (181, **The influence of oxygen upon cultures of the staphylococcus pyogenes aureus, and the oxygen treatment of purulent keratitis in rabbits**) found that oxygen

exerts no disinfecting influence upon cultures of these microbes under the ordinary pressure of the air. On fresh cultures it stimulates the growth of the staphylococcus, but lessens its virulence. Treatment of experimentally produced purulent keratitis by injection of oxygen into the cornea or beneath the conjunctiva showed no real advantage over the usual conservative methods of treatment. HIRSCHMANN.

According to SCHNEIDER (182, **Experimental studies concerning the value of " leucine " in the treatment of infectious inflammations of the conjunctiva**) the normal secretion of the lachrymal gland and conjunctiva contains no bactericide, homolytic, or opsonic substances. After instillation of solutions of silver nitrate, protargol, and zinc sulphate, leucocytes migrate into the conjunctival sac and give up their bactericide material, leucine. The curative action of astringents depends less on their power to form cicatrices or membranes, or their antiseptic power, than on their ability to excite the formation of leucine. The destruction of the infection germs is mainly extracellular in the conjunctival secretion, and is produced by the leucine contained in it rather than by alexine which is ineffective against most exciters of conjunctivitis and comes from the blood in only small quantities after the application of silver and zinc salts.

It appears from the studies of VEDERAME (183, **Experimental studies concerning the action of the bile and its salts upon pneumococci**) that pneumococci, in the condition in which they are met with in the pus of the lachrymal sac and in the vitreous, are dissolved by neither the bile of rabbits, nor by the biliary salts in concentration up to 10% after any practically permissible length of time. Still less is it to be expected that such an effect will be produced on pneumococci in the denser and more resistant corneal tissue. Hence in the treatment of ulcus serpens bile or biliary salts deserve no serious attention. CALDERARO.

JAKAHASHI (184, **Studies concerning the influence exerted upon the germ contents of the conjunctival sac by operations on the globe**) found after operations no increase of the germ contents of the conjunctival sac in 43%, a slight increase in 30%, and a marked increase in 8%, while in the eye not operated on he found a slight increase in 50% and a marked

increase in 50%. It was remarkable that when both eyes of the same patient were bandaged the germ contents of the conjunctival sac of the eye operated on was affected by other and more favorable influences than the other.

WESSELY (185, **Experiments on the growing eye**) reports his observations concerning the development of the eye after various operative interventions. Eyes in which iridectomy has been performed are slightly backward in growth, iridencleisis results in an enlargement of the eye, after posterior sclerotomy there is a slight diminution in the size of the eye. From iridectomy comes coloboma of the lens; from discission of the lens of the new-born rabbit comes traumatic cataract, whence later comes central cataract, while the entire eyeball, as well as the orbit, remains smaller than the control eye. From free discission comes occlusion of the anterior chamber and glaucoma (buphthalmos); a very small iridectomy prevents the further over-enlargement. Tenotomies of the eye muscles produce changes of the refraction and astigmatism.

STANCULEANO and MIHAIL (186, **The pathology of the ophthalmo reaction**) show that the local action of tuberculin is such that the two elementary layers of the conjunctiva, the epithelium and the ground substance, are separated from each other and react specifically. By its effect on the epithelium tuberculin calls forth an excessive reaction, which secretion is specific in the sense that the fluid secreted and accumulated in the vacuoli of the epithelium exerts an attractive power only upon the polynucleated cells. The latter enter the epithelium and are in part subjected to the digestion of the fluid, in part wander to the surface and cause a secretion. This attraction of the polynucleated cells, which is exerted by the specific secretion of the epithelium, increases from the beginning and almost to the end of the ophthalmo-reaction and then ceases, while the polynuclear unfiltration quickly retrogresses. The reaction of the conjunctiva to tuberculin is not a pure lymphocyte reaction, but a mixed reaction, a polynuclear change of longer, and a lymphocyte congestion of shorter, duration.

According to LEBER (187, **Serodiagnostic studies in syphilis and tuberculosis**) the serological demonstration of syphilis by complement fixation in the Wassermann test is a clinically

specific reaction of importance in the diagnosis of infectious eye diseases. A positive reaction is an absolute proof of a manifest or latent infection of syphilis in the organism. Syphilitic products demonstrable by complement fixation form an extraordinarily high percentage in syphilitic eye diseases, even when the infection dates back many years. In congenitally syphilitic eye diseases the serological test gives as high a percentage as in acquired syphilis. The positive reaction alone has full value; it shows, however, only the continued existence of the syphilitic process, it does not indicate its localization. The absence of syphilitic reaction products is no reliable evidence against the existence of a syphilitic infection. In a certain series of cases of surely tuberculous eye diseases antituberculin in the blood serum may be demonstrated by the method of complement fixation, and as this has as yet been found only in the blood serum of persons with tuberculosis its presence justifies the assumption of a tuberculous disease. The absence of an antituberculin reaction is no evidence against tuberculosis; the positive reaction is not a final proof of its presence. A thorough clinical examination must go hand in hand with the serological. A syphilitic and a tuberculous infection may be demonstrated simultaneously; in such cases a clearing up of the etiology may be expected through the results of treatment. The tuberculous origin in a series of cases of chronic iridocyclitis and of disseminated chorioiditis could be shown.

According to SCHUMACHER (188, **The serodiagnosis of syphilis in ophthalmology, together with remarks concerning the relations of tuberculosis to syphilis in eye diseases**) the serodiagnosis is of real value in many doubtful cases of syphilis, not only through the positive reaction, but occasionally through the negative. In hereditary syphilis the percentage of positive reactions in cases that have never had any acute symptoms, or have not had any for a long time, appears to be low. Tuberculosis within the eye may develop on the basis of an inflammation of the eye due to hereditary syphilis. In those cases in which syphilis and tuberculosis come simultaneously into account for the eye disease a combined treatment directed against both diseases would seem to be indicated.

JACKSON'S (189, **Blood pressure and its practical relations**

to ophthalmology) able paper discusses retinal venous and arterial pulsation and their occurrence in disturbances of general circulation and local conditions. He also describes the appearances of the vessels in arteriosclerosis and high blood-pressure, namely changes in the vessel walls, such as thickening, commonly varying, narrowing of the arteries, broadening of the light reflex, tortuosity of the small branches, narrowing or obliteration of veins by crossing arteries, changes of color of the arteries, as well as blurring of the disk. He shows that the usual signs of general arteriosclerosis and high blood-pressure are much less valuable and conclusive than the ophthalmoscopic findings. He closes the paper with mention of eye conditions depending upon blood-pressure changes such as atrophy of the optic nerve from lowered blood-pressure as in severe hemorrhage and the universal association of simple glaucoma with increased tension.

ALLING.

By dark adaptation we understand, according to STARGARDT (190, **Disturbances of dark adaptation**), the adaptation of the eye to such slight light stimuli as are met with in darkened rooms or in the dark. Disturbances of dark adaptation are met with in myopia, mixed astigmatism, medullated nerve fibres, old intraocular hemorrhages, siderosis, albuminuric retinitis, retinitis pigmentosa, detachment of the retina, fresh and old choroiditis, optic neuritis, optic atrophy, glaucoma, retrobulbar diseases of the optic nerve, hemianopsia, and opacities of the refractive media.

According to BIRCH-HIRSCHFELD (191, **The changes in the anterior segment of the eye after frequent irradiation with short-waved light**), short-waved light in an intensity sufficient to produce a well marked electric ophthalmia produces no microscopically demonstrable injuries in either the lens or the retina of the lens-containing eye even when the irradiation has been repeated very frequently and the observation has been continued for a long time. The origin of vernal catarrh is not to be ascribed to the action of the rays, even though much importance may be attributed to it in so far as by its irritative action it may excite a recurrence in an already diseased conjunctiva.

DAVIS (192, **The treatment of the eye when the globe is**

infected, with the object of preventing panophthalmitis) reports upon the favorable action of argyrol injected into the anterior chamber in cases of beginning panophthalmitis. He has observed four cases, one following discission in which $\frac{8}{10}$ vision was finally obtained and three cases of infection after trauma where the argyrol seemed to check the progress of the infection. The solution of argyrol varied in strength from 2 to 20%, and was injected several times in each case. Bacteriological examination was made in only one case, which showed pneumococci. The eye was subsequently removed because of beginning sympathetic trouble.

ALLING.

As ELSCHNIG and ULBRICH (193, **Etiology and prophylaxis of the post-operative inflammations of the eye**) state, post-operative inflammations are caused only in part by infection and are in part due to the trauma of the operation and the remains of cataract that have been left behind. Post-operative infection with morphologically the same exciter may appear as a very slight iritis, or a severe plastic or purulent iridocyclitis with infiltration of the vitreous. The difference depends on the virulence of the infection, the number of germs introduced, and the general predisposition. The most frequent exciters of post-operative infections of the eye are the streptococci and the pneumococci. The infection comes usually from the conjunctival sac; perhaps in a few cases post-operative iritis is due to bacteriæmia. Prevention consists in the detection and elimination of streptococci and pneumococci from the conjunctival sac.

In burns and injuries of the third degree of the skin and mucous membrane of the lids, PFALZ (194, **Treatment and after-treatment of wounds and burns of the eye**) recommends, after removal of necrotic tissue, to cover the involved area with Thiersch's grafts as soon as possible after the separation of the slough rather than to wait for spontaneous cicatrization. In the treatment of fresh burns care should be exercised to limit so far as possible secondary infection. The use of astringents should be limited to the treatment of secondary catarrhs, and their application should be as short as possible. The increase of vessels in the conjunctiva after burns of the second and third degrees are not significant of inflam-

mation, are not accompanied by trouble, and should not be treated.

ELSNER (195, **Clinical and experimental studies concerning the cause of the symptoms of intoxication after the use of atropin in children suffering from diseases of the eye**) observed toxic symptoms after instillation of a ½% solution of atropin into the eyes of several children, and traced them to the hyoscyamin which was mixed with the drug. Care should be taken to use only atropin that is free from the much more poisonous hyoscyamin.

According to POSEY and SAUTER (196, **An ophthalmological phase of the dangers of consanguineous marriages**), statistics show that at least four per cent. of the blind and deaf and seven per cent. of all idiots are the children of consanguineous parentage and that these defects are transmitted through many generations. In view of these facts they emphatically condemn the marriage of cousins. ALLING.

FRAZIER (197, **The surgical aspect of cerebral decompression**) adds to the accumulating weight of opinion that this operation is indicated in papilœdema for the purpose of saving (though it be temporarily) the eyes from blindness and relieving also other symptoms of brain compression. His conclusions from experiments upon animals, however, do not agree altogether with those of Cushing and Bordley, who believe that œdema of the disk is mechanical and entirely due to intracranial pressure. He thinks that other and at present unknown factors are responsible. ALLING.

III.—REMEDIES AND INSTRUMENTS.

198. v. HIPPEL, A. **The therapeutic value of Deutschmann's serum.** *Archiv f. Ophthalmologie*, lxxii., No. 2, p. 301.

199. HAPPE. **Critical considerations of Deutschmann's serum therapy.** *Muenchener med. Wochenschrift*, 1909, No. 33, p. 1662.

200. PIGNATORI. **The organic combinations of silver: protargol, argyrol, and sofol in ocular therapy.** *Annal. di Oftalm.*, xxxviii., Nos. 6, 7.

201. KEY, B. W. **Effects of atoxyl on the eye.** *Penn. Med. Journal*, June, 1909.

202. BRAV, A. **Homatropin poisoning with complete transient aphasia.** *Penn. Med. Journal*, April, 1909.

203. ELLIOT, R. H. **A note on alypin.** *Ophthalmoscope*, Oct., 1909.

204. ROCKLIFFE, W. C. **A new danger arising from the use of plated instruments in ophthalmic operations.** *Ophthalmic Review*, July, 1909.

205. GIFFORD, H. **An instrument for opening from the lachrymal sac directly into the nasal cavity.** *Ophthalmic Record*, Oct., 1909.

206. SPENCER, F. R. **Stitch scissors for eye, ear, nose, and throat.** *Journal A. M. A.*, Nov. 20, 1909.

207. GRIMSDALE, H. **A simple substitute for a rotating prism.** *Ophthalmoscope*, July, 1909.

208. DUANE, A. **A standard test object for determining the near point and the range of accommodation.** *Ophthalmic Record*, July, 1909.

209. OLIVER, C. A. **The requirements and regulation of signalling by colors.** *Ophthalmology*, Oct., 1909.

According to v. HIPPEL (198, **The therapeutic value of Deutschmann's serum**) the value of Deutschmann's serum is proven in infectious corneal ulcers and severe plastic iritis which are not of a syphilitic or a tuberculous nature; in all other inflammations of the eye it fails, at least in doses of from 2 to 4*ccm.*

HAPPE (199, **Critical considerations of Deutschmann's serum therapy**) tested the bactericide value of Deutschmann's serum in experimentally produced infectious diseases of the eye with a completely negative result.

From his numerous experiments PIGNATORI (200, **The organic combinations of silver, protargol, argyrol, and sofol in ocular therapy**) draws the following conclusions: Of these three combinations of silver, protargol paralyzes the movement of the leucocytes for the longest time; sofol is the least poisonous. The strength of the solution of each that can be borne without injury to the vitality of the cell protoplasm is for argyrol 1:200,000, for protargol 1:512,001, for sofol 1:180,850. These solutions are not astringent in the least, so do not act by constriction of the vessels; they are not hæmostatic, but rather exert an anticoagulation effect upon the blood; they are not caustic. Protargol is a good bactericide, argyrol and sofol are less efficient. All possess aseptic power, protargol to a more marked degree. All three call forth an increase of the opsonic index and of opsonin in vitro, sofol most efficiently. Weak solutions of the latter could therefore be used for injections between the corneal lamellæ in cases of ulcers for the purpose of exciting irritation followed by œdema with an escape of plasma and leucocytes. Practically protargol is especially indicated in severe catarrhs with abundant secretion and in purulent conjunctivitis; argyrol and sofol

in the milder forms of conjunctivitis, particularly when complicated with corneal lesions. In hypertrophy of the papillæ silver nitrate must be relied on. CALDERARO.

This drug is employed only to a limited extent, and although it appears from the studies of KEY (201, **Effects of atoxyl on the eye**) to be of therapeutic value it is treacherous on account of its tendency to produce optic atrophy with involvement of the retina. ALLING.

After instillation of homatropin hydrobromate, one-half grain to dram, for eight times, BRAV (202, **Homatropin poisoning with complete transient aphasia**) observed the usual toxic symptoms in cases of idiosyncrasy but also a complete aphasia, lasting seven hours. This may either have been hysterical or toxic from inhibition of the function of the cortical centre of speech. ALLING.

ELLIOT (203, **A note on alypin**). Major Elliot working at Madras gave alypin a trial upon a day when nineteen extractions had to be performed. A 2% solution was used, five instillations being given at intervals of five minutes. This is the plan adopted with cocain, and it has proved perfectly satisfactory. Alypin proved most unsatisfactory, the patients felt severe pain, and there was unusual hemorrhage. The drug dilated the pupil but not so much as cocain. For pterygia operations alypin seemed more suitable, complete anæsthesia being attained, but hemorrhage was more troublesome than with cocain. Adrenaline was used with each drug. The author sees no reason to use alypin instead of cocain.

T. HARRISON BUTLER.

ROCKLIFFE (204, **A new danger arising from the use of plated instruments in ophthalmic operations**). The author lost an eye from panophthalmitis following an extraction. A piece of plating from de Wecker's scissors, measuring 1.5*mm* x 1*mm* was found in the pus which escaped from the eye. He suggests that this accident may account for cases of acute panophthalmitis following extraction which most surgeons can recall, but which they have been unable to explain. The explanation hardly seems reasonable. The panophthalmitis was in an acute stage on the third day. No *sterile* piece of plating introduced into an eye could produce such a result. The explanation of these cases has been

suggested by Axenfeld. The most frequent cause of panophthalmitis is the *pneumococcus*. This according to Axenfeld is common in the mouth, but comparatively infrequent in the conjunctival sac. During an extraction the surgeon is compelled to talk to the patient, and ***unless he wears a mask*** he is spitting bacteria, cocci, and spirillæ into the wound! Let us not deceive ourselves. When we get panophthalmitis we have in some way or other infected the eye. The author does not tell us what organisms were found in the pus. Nevertheless his advice to examine our instruments for loose plating is most valuable. T. HARRISON BUTLER.

GIFFORD (205, **An instrument for opening from the lachrymal sac directly into the nasal cavity**) has devised a rather formidable instrument in the shape of a punch, one arm of which is inserted into the nose; the other, which is long and hollow, is introduced through the sac and is forced through the stricture. The procedure has some obvious disadvantages. ALLING.

SPENCER (206, **Stitch scissors for the eye, ear, nose, and throat**) has invented a pair of scissors, the tip of one blade of which is converted into a hook making it possible more readily to pick up and sever the stitch. ALLING.

GRIMSDALE (207, **A simple substitute for a rotating prism**). The author finds that the method of testing for an error of muscle balance by inserting prisms in the trial frame is not very satisfactory. The rotating prism is heavy and unsuitable for the trial frame. He has designed the following test. A perimeter chart is taken, and a series of vertical and horizontal lines are ruled, passing through the points where the concentric circles intersect the vertical and horizontal meridians. Each 10° interval of the visual field represents the deviation of any unit prism, in the author's practice 1°. Maddox's rod is placed in the posterior cell of the trial frame, and a 9° prism placed in front of it base down. As the prism rotates, the line of light rotates and its position can be referred to the chart. T. HARRISON BUTLER.

DUANE (208 **A standard test object for determining the near point and the range of accommodation**) believes that the fine print usually employed for this purpose is too coarse and has devised a new test object. It consists of a disk 38*mm* in diameter and covered with black velvet to form a dull back-

ground. On the centre of the disk is fixed a white card 3*mm* by 1.25*mm* which is divided longitudinally by a sharp black line 0.2*mm* wide. The near point is measured when the black line is first blurred in approaching it to the eyes. The test is very delicate. ALLING.

OLIVER (209, **The requirements and regulation of signalling by colors**) enters a plea for the standardization of all color signals by means of an international commission. Each vessel should be provided with sample colors so that all materials used in signalling shall be of uniform standard. Studies should be made of the amount of illumination necessary to produce a given brightness for each color used.

ALLING.